THE OPEN UNIVERSITY

Science Foundation Course Units 19 and 20

Evolution by natural selection
Species and population

The Open University

Science Foundation Course Unit 19

EVOLUTION BY NATURAL SELECTION

Prepared by the Science Foundation Course Team

THE OPEN UNIVERSITY PRESS

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The Open University Press
Walton Hall Bletchley Buckinghamshire

First published 1971. Reprinted 1972, 1973

Designed by the Media Development Group of the Open University

Printed in Great Britain by
EYRE AND SPOTTISWOODE LIMITED
AT GROSVENOR PRESS PORTSMOUTH

SBN 335 02033 X

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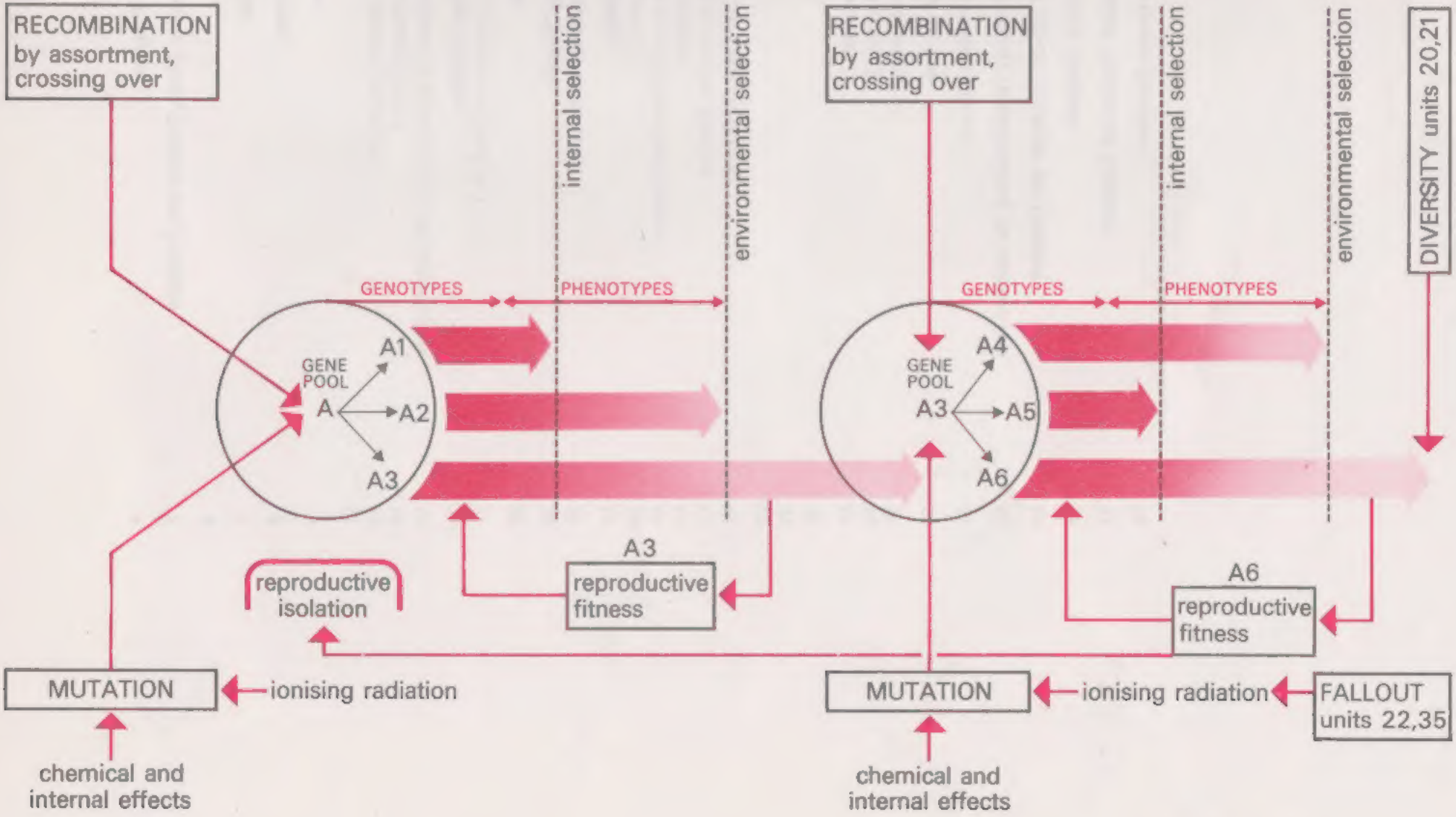
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Table A

List of Scientific Terms, Concepts and Principles used in Unit 19

Taken as pre-requisites			Introduced in this Unit			
1	2		3		4	
Assumed from general knowledge	Introduced in a previous Unit	Unit No.	Developed in this Unit or in its set book(s)	Page No.	Developed in a later Unit	Unit No.
	DNA	13	adaptation	9		
	genetic information	17	clone	11		
	genetic continuity	17	dominant	13		
	mitosis	17	recessive	13		
	chromosomes	17	chromatid	17		
	co-linearity	17	diploid	17		
	enzyme	10	gonads	17		
	polypeptide chain	14	haploid	17		
	nucleotide	14	homologous chromosomes	17		
	gametes	17	meiosis	17		
	phenotype	17	crossing over	22		
	genotype	17	assortment	23		
	phage	17	polyploid	23		
			recombination	23		
			mutation	25		
			population	28		
			evolution	29		
			inheritance of acquired characters	30		
			natural selection	31		
			micro-evolution	36		
			selection	36		
			heterozygous	42		
			homozygous	42		
			speciation	47		
			species	47		
			gene pool	49		
			gene flow	50		
			hybrid	51		
			intelligence quotient	56		

Scientific terms used in this Unit but not listed above are marked thus † and defined in the glossary (*p.* 61).

Objectives

When you have completed this Unit, you should be able to:

1 Define, or recognize the best definitions of, the terms listed in Table A, p. 5 (*SAQs* 1, 2, 3)

2 List, or select correct examples of:

(a) at least three agents which may cause a rise in the mutation rate in a population of living organisms;

(b) at least two examples of the frequency with which particular mutations are known to occur;

(c) two examples of the chemical change which has occurred in a phenotype as a result of a particular mutation of the genotype;

(d) three examples of the sort of changes in the structure of the DNA or chromosome which might constitute a mutation;

(e) four examples of adaptation in a population arising from selection;

(f) one example of the effect of conflicting selection pressures on the frequency of a gene in a population;

(g) at least one item of evidence that selection is acting on the human gene pool at the present time. (*Text Question* (*TQ*), *SAQs* 4, 7)

3 (a) Draw or select annotated diagrams to show the essential differences in the behaviour and distribution of the chromosomes in meiotic and mitotic cell division.

(b) Interpret experimental data on the redistribution of characteristics following sexual reproduction, in terms of the behaviour of the chromosomes. (*TQ*)

4 Discuss in 500 words the potential evolutionary advantages to a species of sexual reproduction. (*SAQ* 4)

5 Given appropriate data, design simple experiments to:

(a) determine whether a variant in a population is phenotypic or genotypic;

(b) show whether a particular mutation occurs spontaneously or as a consequence of some specific action on the genotype by the environment;

(c) determine whether two characteristics are controlled by genes on the same or different chromosomes. (*TQ*)

6 To select and interpret data which give support for the hypothesis that under some circumstances natural selection is acting to favour stability and under others to favour change in a population. (*CMA*)

7 (a) To recognize the most important factors involved in distinguishing species. (*SAQ* 8)

(b) To recognize the circumstances under which new races may be formed, and from them, new species. (*Unit* 21, *section* 21.1)

8 By means of an essay of 1 000 words, or a suitable essay plan:

(a) outline Darwin's theory of evolution and compare its ability to explain the living world with that of earlier theories;

(b) construct an argument to support the idea that the evolution of species, and their adaptation to their environment, can be explained solely on the basis of selection acting on random variation;

(c) make a reasoned case for or against the statement 'that change in the human genotype is likely to be an important factor in future human evolution'. (*SAQ* 9)

9 Apply principles expanded in this and earlier Units to totally new (given) situations to which they are relevant. (*CMA*)

General Aims

1 To show how genetic variations may arise within a species or a population, and how this variation, coupled with the over production of offspring, can lead to a change in the prevalent genotype of that species or population.

2 To consider species and speciation in terms of the theory of natural selection.

3 To induce the reader to consider a number of practical problems in the light of current views on the action of selection on populations.

Study Comment

1 It is important that before you read this Unit, you should consider the objectives. As with the other Units in the course, it is only by looking at the objectives and the *SAQs* that you can know what we hope you will gain from it. They should give you a clear idea of what facts, ideas and principles we expect you to retain after completing it.

2 Brief summaries have been provided at the end of the section on mutation (19.2.1), the section on meiosis (19.2.3) and at the end of the Unit. You are asked to make your own very brief summaries of what you consider to be the important points, at the end of the sections on evolutionary theories (19.5), micro-evolution and adaptation (19.6) and speciation and gene pools (19.7). We have indicated what we consider these to be, and we hope there will be a reasonable amount of agreement between your version, our version and the objectives!

3 Quite early in section 19.2.3, when the basic mechanics of meiotic cell division have been introduced, you are asked to do the first parts of the Home Experiment, which involves the use of photographs rather than actual live material. We hope that this will satisfy you that the main points we make about meiosis are in fact true.

The set reading for this Unit is a paper by Dr. N. G. Smith on the reproductive isolation of various species of Arctic gulls, and you should read it when you have completed the sections on speciation and the isolation of gene pools.

4 If you care to pursue the topics raised in this Unit further, we recommend that you read T. Dobzhansky's *Evolution, Genetics and Man* (Chapman and Hall, Wiley, 1955). On a rather narrower front, *Mankind Evolving*, by the same author (Bantam Books, 1970), is a most interesting book, well worth your attention.

19.1 Introduction

If you think back over the last two Units, 17 and 18, you will be aware of a paradox. Unit 18, particularly the television component of it, gave you some indication of the tremendous diversity of living organisms. There are indeed well over a million and a half distinct types, their bodily form ranging from the bacterial to the elephantine. Yet the message emerging from Unit 17 was one of continuity and sameness. The structure and function of a cell are determined largely by its proteins, and these depend on genetic factors, in fact on the precise sequence of bases in the DNA molecule. Mitosis, the process by which cells multiply, preserves this sequence. So the continuity of form and function conferred on the cell or organism would seem to be potentially everlasting, yet all around us we see diversity.

How has this arisen?

If we say that in the process of cell reproduction 'like begets like', it might seem reasonable to assume that all living organisms must have been present from the start of life. This indeed is the view held by Aristotle and Linnaeus among others. It requires that all organisms arose by a process of 'Special Creation', being produced, in unalterable form, by a Creator. Early versions of this theory assumed that they were all created together, at a time known as the Creation. After the discovery of the fossilized remains of hitherto unknown species in rocks known to be of different ages, it was suggested that creation was a sequential process, with intervals of catastrophe wiping out all life, followed by re-creation – a doctrine known as catastrophism.

'Special Creation'

catastrophism

An alternative view is that life may have arisen as just one or a few types of simple organism and that all the present and extinct forms have evolved from these. In this event it will be likely that such evolution will still be continuing. This general theory has, for nearly 100 years, had the support of almost all serious scientists. The ideas of 'Special Creation' are of course impossible to disprove; they are not testable scientific theories, but articles of faith. In this Unit, we are not setting out to prove that biological evolution has occurred, nor to examine its philosophical implications. Its occurrence is generally recognized, so here we will consider only the mechanism by which it has occurred.

However, there is something perhaps even more striking than the great diversity which living organisms show, and that is the extent to which they are precisely adapted to fit their environments. Any detailed study of an organism reveals the truly amazing degree to which its form, physiology, biochemistry and behaviour are exactly suited to the way it lives.

adaptation

For example, a plant may achieve sexual reproduction by producing a flower which is shaped and coloured in such a way as to lure just one particular species of insect. This insect must be flying at the time of year that the plant has ripe pollen, and the behaviour of the insect and the structure of the flower must be precisely co-ordinated if the method is to work. If the adaptation is not correct, it will be useless.

Fish living in cold water will require enzyme systems able to maintain all aspects of life at a low temperature, whereas the enzymes of a fish living in a tropical swamp must achieve the same results in water nearly as warm as a mammal's bloodstream. The tropical fish will perish from cold in a trout stream, as its enzymes will not be able to maintain essential functions

at a sufficiently fast rate. Equally, a trout will die in a tropical swamp, but in this case because there will be insufficient oxygen dissolved in the warm, stagnant water (its respiratory system is adapted to work in cold, but well-oxygenated water). The complex nature of these adaptations seems at first sight little short of miraculous, and it is hardly surprising that adaptation has often been taken as evidence of divine planning. Any scientific theory of evolution must therefore account satisfactorily for this fundamental phenomenon, exhibited by all living things.

No form of evolution can occur unless there is genetic change or variation, as well as the continuity discussed in Unit 17; thus the first section of this Unit deals with variation. We then consider the mechanism by which variation is exploited, and how this may lead to new species, finally, very briefly, we consider in what directions evolution appears to be going at the moment.

19.2 Variation

19.2.1 Variation caused by mutation

mitotis

So far you have considered only one reproductive process, *mitosis* (Unit 17). As a prelude to mitosis each of the parental chromosomes duplicates to form two identical copies, or *chromatids*, one going to each daughter cell. So the daughter cells should be genetically identical, to each other and to the cell from which they arose. Many unicellular organisms, such as *Euglena* (TV programme of Unit 18), reproduce entirely by mitosis, or, as in the case of bacteria, by a similar form of 'simple division' not usually called mitosis because of the different arrangement of the genetic material within the cells. A succession of organisms produced mitotically from a single ancestor is sometimes referred to as a *clone*. The term can be applied to cells as well as to organisms, for example the cartilage cells mentioned in Unit 17. One would expect all members of a clone to be genetically identical, and so to be *potentially* identical in all respects—appearance, growth, metabolism etc.

clone

genetically identical individuals

In fact, the adults of genetically identical individuals may not be identical in all respects because they may have received rather different treatment by the environment. For example, human 'identical' twins are rarely quite indistinguishable, although they share the same patterns of DNA. One of them may have been ill when a baby and be smaller, or he may have a scar on his nose. His behaviour may be rather different: he may be afraid of horses, having had his nose bitten by one.

However, unicellular organisms can be grown in a laboratory under almost standard conditions, so that they receive substantially the same treatment from the environment. For example, bacteria may be cultured on a specially prepared nutrient jelly in covered glass dishes in an incubator. Careful mixing of the jelly ensures that they all receive the same foodstuffs in the same concentrations. The incubator ensures that the temperature, light and humidity are constant for all the individuals at all stages of their growth. If the cultures are set up under sterile conditions, the bacteria will only have each other for company, there will be no other organisms present which might affect some individuals and not others. Under these conditions you might expect that all the individuals of a clone would be indistinguishable from one another. There are many ways in which this may be tested. One such way is to take *Escherichia coli* (*E. coli*), a common bacterium from the gut of man and many other mammals, and grow a clone at 35° C in the standard culture dish known as a Petri dish. Under optimum conditions these organisms will divide every 20–30 minutes and they will be well distributed across the dish by the time they number a few billion (say 3×10^{10}). When examined microscopically they will appear identical.

Another way to examine them, however, would be to add a suspension of T4 bacteriophage to the cultures.

From what you know from Unit 17, what would you expect to happen to the culture?

The phage will attack the bacteria and destroy the culture.

When examined by eye, the growing colony will appear as a large stain, which spreads across the jelly from the point of inoculation. When the phage is added, the colony will apparently be wiped out. But within a few hours one or a small number of spots will appear on the plate and begin to spread. These represent the start of new colonies, arising from one or more bacteria which were not killed by the phage. The resistance of the survivors is clearly heritable, for by the time they can be seen with the naked eye the new colonies number thousands of individuals, all growing in a medium containing large numbers of T4 phage.

heritable resistance

Obviously you would have been mistaken to think that all the individuals of the original clone were identical; a very few of them, perhaps one in every hundred million, must have possessed a striking difference—immunity to attack by T4 phage. Similar experiments using the antibiotic streptomycin instead of phage, again show that the bacteria are not all identical; in this case a few individuals are resistant to the antibiotic. These experiments raise a number of interesting points, and will be mentioned again on pp. 33–4.

All the bacteria are descended from the same ancestor by mitotic division, yet they are not all identical. What has happened? Somewhere in the sequence of divisions a sudden, sharp change has occurred: a genotype which does not confer immunity against T4 phage or streptomycin has changed to one which does. The change may be said to be sudden in this case because there is no evidence for any state of partial or transient resistance which precedes full resistance. A change of this type to the genotype is called a *mutation*. Precisely what a mutation involves chemically is now becoming clear in a few instances. An enormous amount of research into the genetics of *E. coli* has been performed in the last twelve years, indeed a fair amount of the total knowledge of chemical genetics is based on the genetics of this bacterium and of the phages which attack it. (In the discussion on co-linearity in Unit 17, the examples used were in fact mutants of the T4 phage.)

mutation

From what was said in Unit 17, it is clear that a change in the genotype will almost certainly mean a change in the configuration of the nucleic acid. Obviously, a large change in the template is likely to have a drastic effect on the phenotype; it is, however, important to try and judge what is the basic or minimum unit of change that could be called a mutation. If we accept the definition of a gene as being that genetic unit which controls the production of one polypeptide chain, then clearly a mutation may involve only one gene, or even a small part of one gene.

gene

This is of course implied by the theory of the 'triplet code' but direct evidence has been hard to come by. If the theory is correct—and the evidence for co-linearity goes far to support it—a change in one nucleotide may result in the incorporation of a different amino acid into the protein finally produced, and this in turn may profoundly affect the nature and function of that protein, and thus the cell.

triplet code

For example, some mutants of *E. coli* cannot grow unless the medium they are in contains the amino acid tryptophan. The original, or 'wild-type', can manufacture its own tryptophan, provided that the amino acid serine is present. Mutants that cannot do this turn out to have a defect in the protein of the enzyme that is used for the conversion. In one case,

the defect is caused by the replacement of one molecule of the amino acid glycine by a molecule of arginine at a particular point in the protein. Thus a functional change in an organism has been shown to be due to the change of a single amino acid in a protein made up of 267 amino acids.

The speed with which *E. coli* can be bred made it possible for workers in Europe and America to identify several other changes in the same enzyme, and relate them to specific changes in the bacterial DNA (Fig. 1). It emerged from this work that not only do the positions of the mutations on the DNA correspond with the sites of the changed amino acids on the polypeptide chain (providing further evidence of co-linearity) but that by comparing these amino acids with their known codons (Unit 17) it can be deduced that each mutation *could* indeed result from a change in a single nucleotide. This would be in line with predictions made from the 'triplet code' theory. It is a remarkable thought that a visible, functional change in a whole organism may result from the changing of a single nucleotide on a strand of DNA.

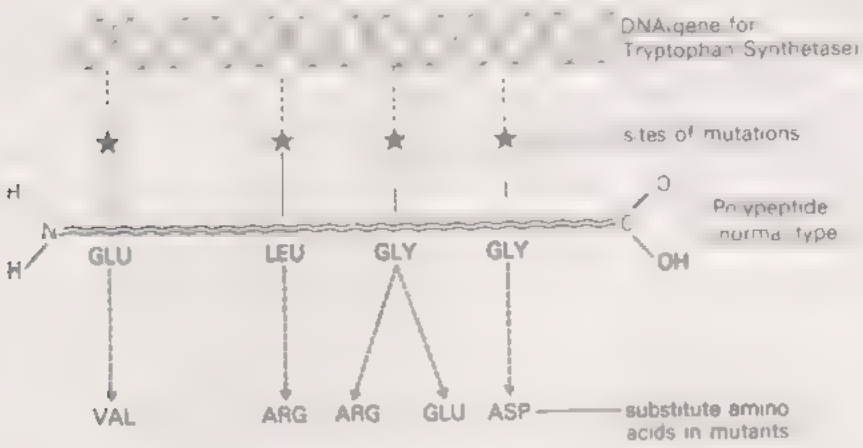


Figure 1 Showing co-linearity of the sites of changes on the bacterial DNA with those of substituted amino acids in the protein of the enzyme tryptophan synthetase.

But, although this work gave the first evidence that a mutation might involve as little as a single nucleotide on a strand of DNA, the first demonstration that a visible change in the phenotype could be traced to just one amino-acid change in a protein was achieved earlier than this, by Ingram in 1957. In this case, the discovery did not lend itself so well to fundamental investigation, but in many ways was even more interesting. Since about 1950, a good deal of interest had been taken in a disease known as sickle-cell anaemia. This is an often fatal 'anaemia' (anaemia in the sense that it is a failure of the red blood cells) which is relatively common in Africans, particularly West Africans. Whenever the blood cells of a victim encounter a low level of oxygen, as in the tissues or venous system, they are liable to collapse into a sickle shape and may form blockages and other complications in the blood vessels.

sickle-cell anaemia

It was found that this disease is inherited, as a single mutant *recessive gene**. The consequence of a child inheriting the sickling gene from both parents is that it has only a 20 per cent chance of surviving to maturity.

* *The term recessive in a genetic context means that the character determined by the gene —in this case the disease—only shows itself in the phenotype if it appears in the relevant chromosomes from both parents. If it appears in just one, with the normal gene opposite, it does not affect the phenotype. The character determined by the normal gene is then said to be* dominant.

Where an identical gene has been inherited from both parents the individual is said to be homozygous *for this gene. Where the genes on the corresponding points of the relevant chromosomes are not identical, for example where a mutant is opposite to the normal gene, the individual is said to be* heterozygous *for this gene. Thus a sufferer from sickle-cell anaemia must be homozygous for the sickling gene, as it is recessive.*

It was discovered in 1949 that the difference between sickling and normal red blood cells was in the haemoglobin they contained. Haemoglobin (Hb) molecules are constructed of two different types of polypeptide chains, α and β. Each chain contains about 150 amino acids. Two of each type of chain are present in each haemoglobin molecule.

The manufacture of the α chain is controlled by a different gene from that of the β chain, so that a single mutation will affect either one pair of chains or the other, not both. The haemoglobin in the sickling cells is known as Haemoglobin S (Hb.S). Between 1956–59, Ingram was able to show that the α chains of Hb S were perfectly normal, as indeed were the β chains except for one amino acid. In position 6 on the chain, normal Hb has glutamic acid, but in Hb S it has been replaced by valine. Thus, the only difference in the primary structure of a molecule with a total of nearly 600 amino acids (4 chains of 150) is that two glutamic acid molecules have been replaced by two molecules of valine (Fig. 2). Yet this difference has killed tens of thousands of people. It seems that the small difference produced by the substitution of two valines for the two glutamic acid molecules normally present, results in profound changes in the folding of the molecule, thus changing its shape. This in turn affects the behaviour of the molecule, particularly its solubility, and seems to result in the Hb molecules clumping together to form long helices when they lose oxygen to the tissues. Thus, apart from the dangers mentioned above, the haemoglobin becomes very inefficient as an oxygen carrier.

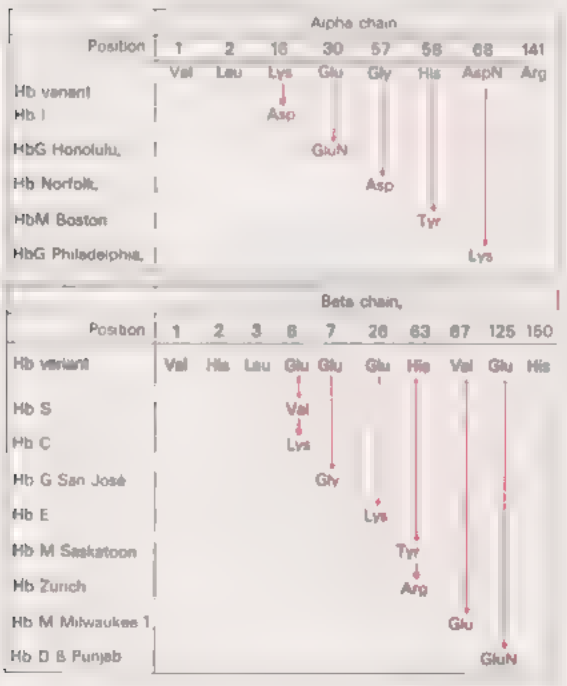


***Figure 2** Positions of substituted amino acids in various mutant forms of haemoglobin.*

The existence of this disease has other implications which will be explained in section 19.6.4, but at this point Hb.S is interesting as the first example of a single gene mutation being shown to affect just one amino acid of a polypeptide chain. Since then other mutated forms of Hb have also been shown to differ by just one amino acid (Fig. 2).

Causes of mutation

It seems that anything which changes the sequence of nucleotides along the DNA molecule could cause a mutation. This change may be merely the substitution of one nucleotide for another, as in the cases considered above, or it may involve several nucleotides. It may also be caused by the deletion of one or more from the sequence altogether, or the addition of completely new ones. Some mutations can certainly be shown to be due to additions or deletions, but it seems that this generally results in the production of a completely non-functional protein.

mutation and chemicals

Mutations can be produced experimentally in the laboratory, by using various different agents. For example, a large number of *chemicals* can be shown to induce mutation. (The actions of some of them are discussed in the black-page Appendix to this section.) They may act to cause deletions, or simply substitutions of single bases.

mutation and temperature

Rather surprisingly, the *temperature* at which some animals, such as the fruit-fly *Drosophila* (film strip 19 20a), are living may have an effect: as the temperature of their environment rises, so does the rate at which mutations occur. This is probably not a relevant factor in animals that maintain a nearly constant body temperature.

mutation and ionizing radiation

Another factor that may induce mutation is the absorption of *ionizing radiation* such as X-rays or accelerated particles from a radioactive source. Irradiation of tissues may result in the breaking of some of the bonds within the DNA. When these are made good, substitutions may occur. This connection between radiation and mutation has led to controversy over the release into the environment of radioactive matter by military and commercial undertakings. You can find more detail of all three of these causes of mutation in Appendix 2 (Black).

Mutation rates

There is, as yet, insufficient evidence to say precisely what causes most naturally occurring mutations. It seems likely, however, that the main cause is chemical. Background radiation is too slight under normal conditions to account for all of the observed mutation rate (at least that observed in *Drosophila*). Also, as it has a cumulative effect, a human with a generation time of nearly 30 years would be expected to accumulate some 360 times as many radiation-induced mutations as *Drosophila*, with a generation time of only four weeks. However, the overall mutation rate in humans seems to be only a little higher than that of *Drosophila*.

In spite of the general state of ignorance about the natural causes of mutation, there is quite a lot of information about the mutation rates of particular genes.

The rate of mutation to T4 phage resistance in *E. coli* is of the order of one in every hundred million, i.e. the chance of it occurring in any one bacterium is 10^{-8}. It has been estimated that in the T4 phage itself the average rate of detectable mutation is 10^{-6} per gene. However there is no reason to suppose that all genes within an organism tend to mutate with the same frequency. Indeed it can be shown in several organisms, including the T4 phage, that there are 'hot spots', i.e. some genes or groups of nucleotides within genes that mutate far more frequently than others.

In maize plants, which live for just one season, seven genes have been compared for frequency of mutation. The rates varied from 492 per million for the colour factor 'R', to less than one per million for the factor 'Wx' (waxy seeds).

Table 1 Mutations observed in seven genes of maize

Gene	*Individuals examined*	*Mutations observed*	*Mutations per 1 000 000 individuals*
R (colour factor)	554 786	273	492
I (colour inhibition)	265 391	28	106
P_1 (purple colour)	647 102	7	11
Su (sugar)	1 678 736	4	2.4
Y (yellow seeds)	1 745 280	4	2.2
Sh (shrunken seeds)	2 469 285	3	1.2
Wx (waxy seeds)	1 503 744	0	0

In humans, mutations to the gene causing haemophilia, the disease in which blood clotting is impaired, arise 20 to 30 times in every million individuals. The mutation responsible for producing achondroplastic dwarfism† apparently occurs more often—50–100 times per million. Whilst this is within the range mentioned above for maize, it is much higher than that for the bacteria and phages, with 30-minute generation times. However, comparisons between such different organisms are rather hard to interpret in the present state of knowledge.

19.2.2 Summary of section 19.2.1

1 The occurrence of mutations can be demonstrated in a number of ways, one of which is to grow a clone of normal *E. coli*, and subject it to massive attack by T4 bacteriophage. If resistant individuals have arisen by mutation, these will found new colonies, which can be counted.

2 Several other mutants of *E. coli* cannot synthesize tryptophan from serine. It is known that the only deficiency in the bacterium is the replacement of one amino acid in an enzyme by another one, but this is enough to make an important change in the bacterium. The points on the bacterial DNA where the mutations have occurred have been identified, and in at least one case the mutation may involve no more than one nucleotide of the DNA.

3 The disease 'sickle-cell anaemia' is known to be caused by a single, recessive gene. The only difference between normal haemoglobin and the mutant form lies in the replacement of one amino acid by another in two of the four polypeptide chains making up the molecule.

4 Three of the factors which may cause mutation are listed, and the rate at which some particular genes are known to mutate are given.

5 From the above, we can say that mutations occur, that the frequency with which some of them occur is known, that some of the agents which induce them are known, and that in at least one case there is evidence that only one nucleotide is involved. In at least two cases, the detectable changes produced in the body of an organism can be attributed to the replacement of just one amino acid of a polypeptide chain.

19.2.3 Variation caused by meiosis and sexual reproduction

Mitosis is probably the simplest way in which a cell or organism may reproduce itself, but many unicellular organisms (e.g. *Paramoecium* which you saw in the TV programme of Unit 18), and most higher organisms, indulge in a more elaborate process which gives rise to a great variation between the genotypes of individuals within a population. This process

involves the combination of genetic material from two individuals, and is well known under the name of sexual reproduction. It is obvious that this type of reproduction will not produce offspring identical to one parent, but ones which are genetic mixtures of both the parents. However, the amount of variation produced is actually very much more than would be achieved by a simple blending of parental characters, and it is worthwhile examining the process in a little more detail.

gamete

In animals, specialized reproductive cells, called *gametes* (the egg and sperm cells) are produced in the *gonads* (called ovaries and testes) by each parent. These fuse, in the process of fertilization, to form a *zygote*, which has a single nucleus containing the genetic material from the two gametes. The zygote then begins to divide, mitotically, to grow into the new organism. In plants, gamete formation also occurs, but follows a rather different pattern.

zygote

meiosis

The process by which the gametes are produced in the gonads is not mitosis, but a different form of cell division called *meiosis*. Clearly the gametes may have to be specialized in various different ways, for example they may have to be motile, like most sperm, or be able to withstand the rigours of the outside world for a considerable period of time. But one specialization they must all have in common is that they shall carry only half the normal (or *diploid*) chromosome number of the adult, and for this reason they are said to be *haploid*. Thus, in man, the eggs or sperm will have 23 chromosomes instead of 46 as there are in the adult; in *Drosophila* 4 instead of 8, in maize 10 instead of 20. The significance of this is obvious. If the gametes contained the full chromosome number, when they fused to form a zygote, the latter would have double the chromosome number of the parents, and so on at each generation. Not only would this not produce the genetic continuity stressed in Unit 17, but in a few generations the organism would soon consist of nothing but chromosomes.

haploid

Thus in one sense, meiosis is merely a specialized form of mitosis that reduces the chromosome number of the daughter cells by half, and does so in a very simple manner. In a diploid organism the chromosomes are in *homologous pairs*, that is to say there are two similar chromosomes in the nucleus, containing genes determining the same proteins (and thus phenotypic† characters), one inherited from each parent.

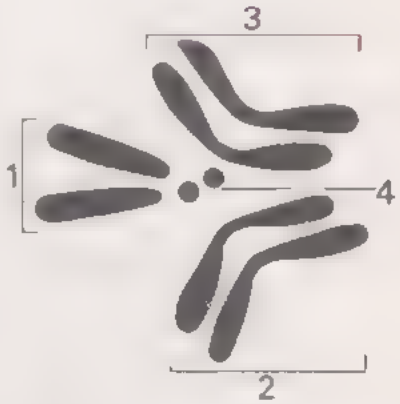


Figure 3 The four homologous pairs of chromosomes in Drosophila.

Meiosis in Drosophila

An example of a diploid organism much used in genetical research is the fruit-fly *Drosophila*, mentioned above. This animal is shown in film strip 19, 20(a) and in Figure 8, p. 21. It normally has eight chromosomes, four homologous pairs (Fig. 3).

You will recall from Unit 17 that, in simple cell division (mitosis), these chromosomes will line up across the spindle† in a single plane, and it can be shown that each chromosome has replicated into two identical chromatids, one of each being later drawn into each daughter cell. Thus, in the case of *Drosophila*, each daughter cell will have one chromatid from each of the eight chromosomes, making up the four homologous pairs. In the early stages of meiosis, on the other hand, the two members of each of the homologous pairs of chromosomes line up across the spindle, i.e. in the equivalent position occupied by the two chromatids of one chromosome in mitosis (Fig. 4, p. 18).

Note. Figures 4–6 are highly stylized diagrams intended to clarify the steps in the process. In the film strip and appendix you will find a series of actual photomicrographs. These will show you the type of material from which we obtain the information used here.

In each homologous pair, the chromosomes are associating very closely.

Figure 4 The four homologous pairs of chromosomes arranged across the spindle at the start of the first meiotic division in ♂ (male) Drosophila.

Consider just one pair for a moment. At this point both chromosomes can be seen to have replicated into two chromatids. The chromosomes consisting of pairs of identical chromatids, are drawn away from each other, one pair going to each side of the cell, just as the single chromatids are in mitosis. Thus, when the *first meiotic division* is complete, the two daughter cells each have only one chromosome (that is, two chromatids) from the original homologous pair (Fig. 5).

first meiotic division

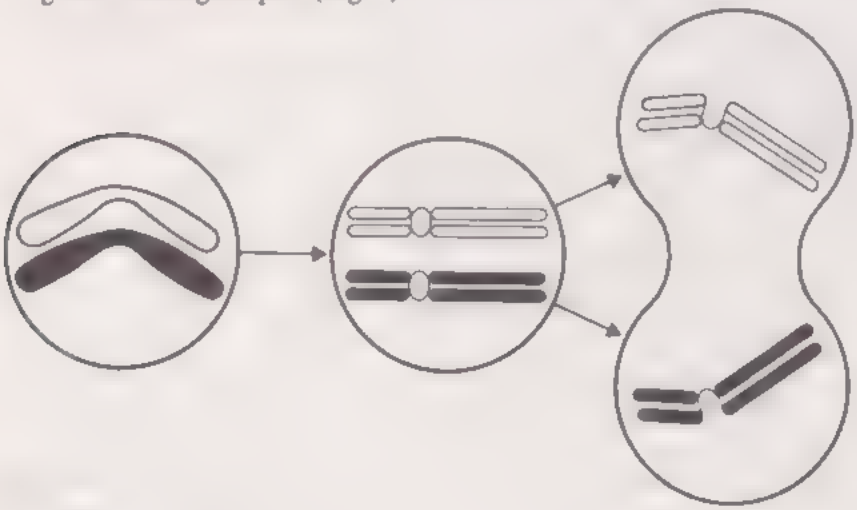


Figure 5 First meiotic division, illustrating the behaviour of only one pair of homologous chromosomes in ♂ Drosophila.

This is promptly followed by a division in each of the daughter cells which closely resembles a normal mitotic division although, of course, with only half the original number of chromosomes. The chromosomes lie singly across the spindle, and one chromatid from each is then drawn into each daughter cell. This completes the meiotic division, in which four sperm have been formed from the original cell, each with a chromatid from only one chromosome of each of the four homologous pairs (Fig. 6).

second meiotic division

As Figure 6 indicates, this process will give rise to four haploid sperm. Remember that one chromosome of a homologous pair is paternal in origin and one is maternal. So, if there were in fact just one pair of chromosomes involved, as in the figure, the sperm would contain genetic material either from the maternal or from the paternal side of the parent cell, but not mixtures of both, i.e. they would resemble genetically one or other of the two grandparents' gametes, which fused to form the genotype of the parental cell.

There are, however, *four* pairs of homologous chromosomes, so each sperm will contain *four* chromatids derived from those original eight chromosomes.

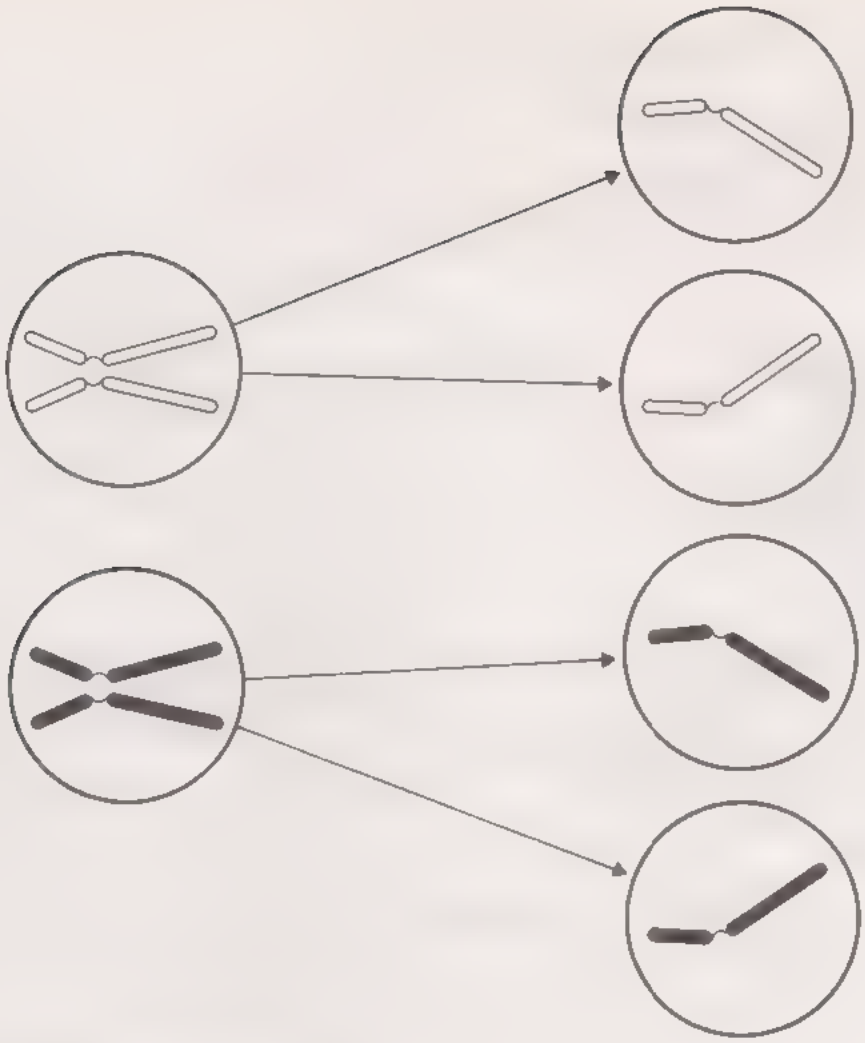


Figure 6 Second meiotic division.

From the information given you here, can you calculate how many different genotypes (disregarding possible mutations, etc.) you might expect to find among the sperm arising from one cell by meiosis?

No, you cannot.
If the homologous chromosomes all pair the same 'way up', relative to one another, across the spindle, then there will be only two different genotypes just as with the single pair of chromosomes. If they do not, then there will be a greater variety of genotypes

To calculate how many different genotypes to expect in *Drosophila* sperm, you must first do part 1 of this Unit's Home Experiment. Just looking at photographs of the chromosomes of a cell during meiosis, as you can do in the film strip 19 20(a), 6, 7, 8, will not give you the answer. You cannot tell whether one of a pair of homologous chromosomes is of maternal or paternal origin from its appearance. It is therefore necessary to infer the answer to the above question by indirect means.

Unfortunately, it has not been possible to send you actual cultures of the organism we have chosen, so instead we grew them and photographed them in the Open University's laboratories. You can see in these photographs what you would have seen through the microscope.

Now do part 1 of the Home Experiment

From the photographs in part 1 of the Home Experiment, you will have deduced that it is purely a matter of chance which way up the homologous pairs lie across the spindle.

You can therefore now answer the question: 'how many different genotypes will be represented in the sperm of *Drosophila*?' If you have any difficulty, you can draw up a small table.

See Answer 1, p. 76.

An example of the actual effect this kind of redistribution may have on the genotypes carried by the sperm is given in the answer to the next question

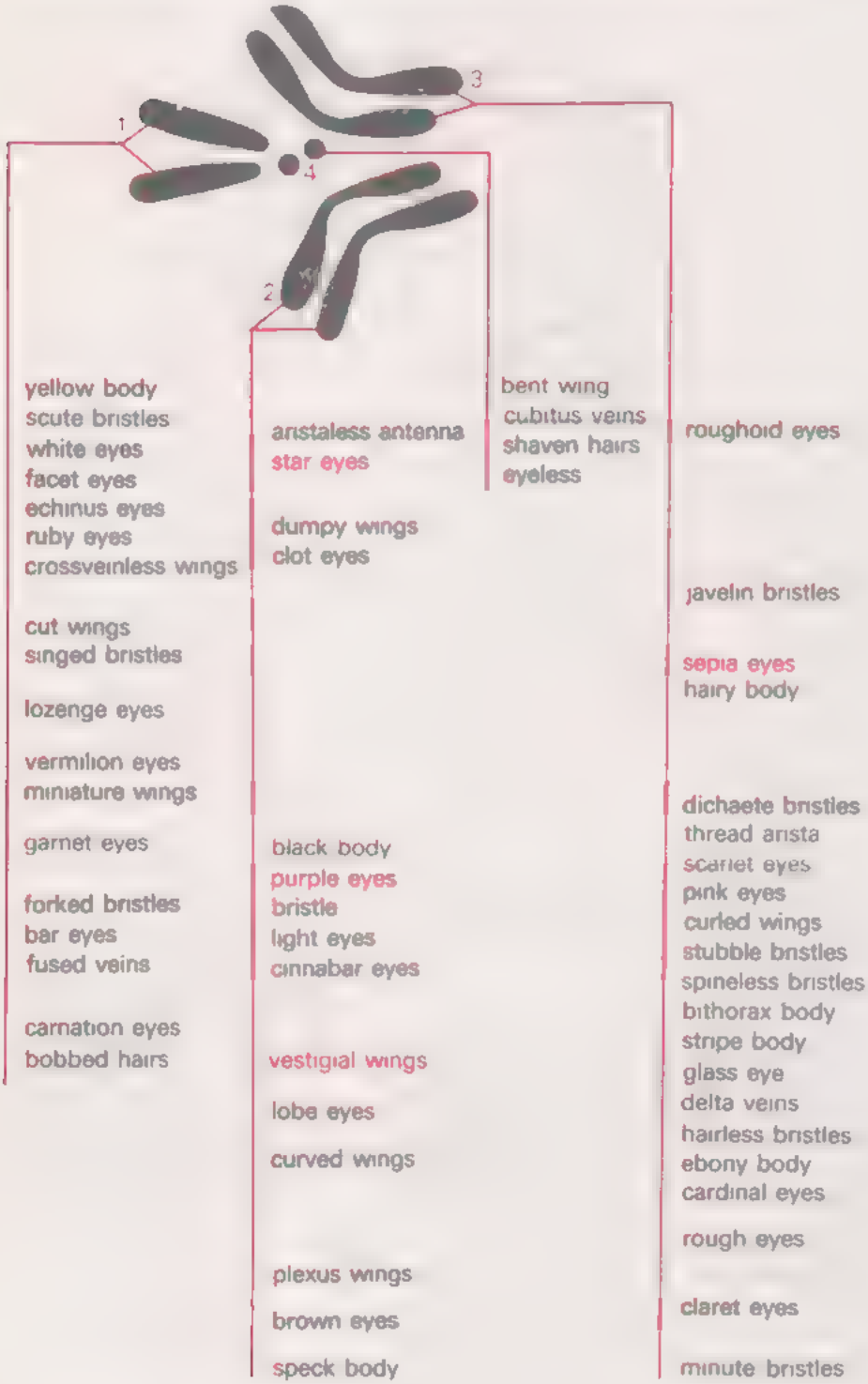


Figure 7 Relative positions of some of the genes carried on the chromosomes of Drosophila melanogaster.

Figure 7 shows the relative positions of some of the more important genes on the chromosomes of *Drosophila melanogaster*. (You are not required to memorize any of these; the figure is given for illustration only.)

In the Home Experiment, the orientation of the chromosomes could only be seen to determine the sequence of spores in the ascus of *Sordaria*. However, using the information in Figure 7, we can see some of the more important genetic effects that this type of redistribution may have. For example, on chromosome 2 there appears the gene for 'vestigial wings'. This is a recessive character, so that if the adult shows it, it means that the gene must appear in both the homologous chromosomes of pair no. 2. It is obvious then that it will appear in *all* the sperm of a male with vestigial wings. If, however, the adult has normal wings, it may be that it has one 'normal' gene and one 'vestigial' gene, the normal one being dominant. In this case, half the sperm would be expected to carry the normal gene and half the vestigial. If we take the same situation with another recessive gene on a different chromosome, say the gene for 'sepia eyes' on chromosome no. 3, the same will apply.

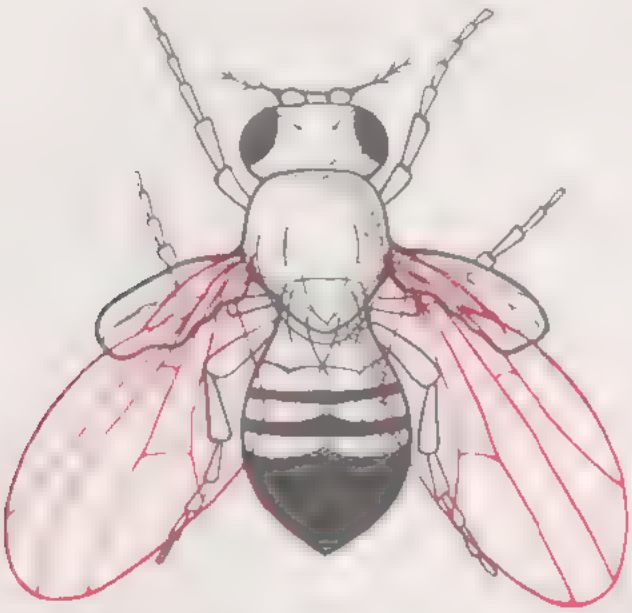


Figure 8 Drosophila *with 'normal' and 'vestigial' wings.*

Thus, if we have a male with normal wings and normal eyes, carrying a recessive gene for vestigial wings and one for sepia eyes, how many different genotypes in the sperm would you expect with regard to these two characters?

See Answer 2, p. 76.

In fact, it is clear that either of the genes for the one character is equally likely to find itself in a sperm with either of the genes for the other character, if it is carried on a different chromosome. It follows, therefore, that these two characters are being distributed, or 'assorted', independently of one another.

independent distribution of character

This will not, of course, be the case where two characters are carried on the *same* pair of chromosomes. For example, the recessive gene for 'purple eyes' is carried on the same pair (no. 2) as that for 'vestigial wings'. There can therefore only be *two* genotypes for these characters among the sperm. Precisely what they are will depend on whether the 'purple eyes' recessive is on the same member of the pair as the 'normal wing'.

At this point you should do the second part of the Home Experiment, if you have not already done so.

Crossing over

You will have seen from your examination of spore formation in *Sordaria* in the Home Experiment that in fact there is greater recombination of characters than can be explained merely by the random arrangement of the homologous chromosome pairs.

You have seen evidence that the two members of each pair may exchange whole lengths of material (part 3 of the Home Experiment) between the time they replicate into chromatids and the time they pull apart. This process, called 'crossing over' is very general indeed, and occurs during meiosis in the male and female of almost all other organisms, and the female of *Drosophila* (Fig 9). The case we have been considering above is exceptional in that crossing over does *not* occur. Thus, if we consider the same two recessive characters ('vestigial wing' and 'purple eyes') during *egg* production, it can be shown that about 15 per cent of the gametes no longer show the parental association. If we take two genes which are further apart on the chromosome, say 'vestigial wing' and 'star eyes', this percentage rises, indicating that the further apart the two genes are on the chromosome, the more likely they are to get separated by crossing over.

crossing over

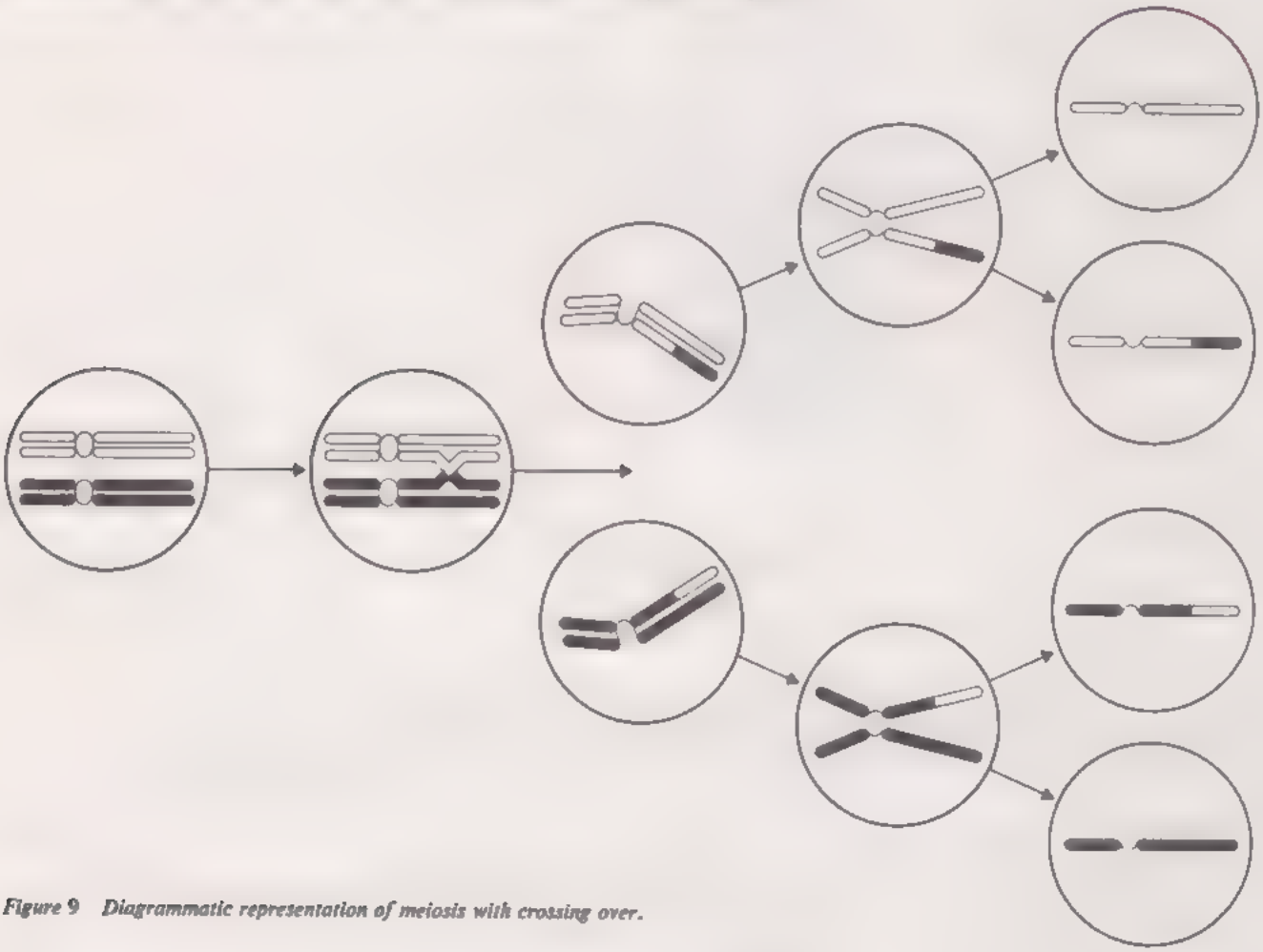


Figure 9 Diagrammatic representation of meiosis with crossing over.

Crossing over only occurs between two of the four chromatids at any one chiasma (see Home Experiment Notes, part 2) However, other chiasmata may form further along the same chromatid, or between the other two chromatids, forming double cross overs (Fig 10). Sometimes, as you have seen, three out of the four chromatids may be involved, one crossing once with one opposite member and, further down, with the other.

The number of cross overs varies very much between species, and between different chromosomes of the same species. An average figure would be two or three chiasmata formed at each pairing of homologous chromosomes.

From this you can see that meiosis provides a very great source of genetic variation, partly due to the assortment of maternally and paternally derived chromosomes, caused by the random orientation of the homologous pairs before division, and partly because of the exchange between chromatids in crossing over.

The extent of recombination

recombination

There is effectually no limit for the number of possible recombinations in the genotype that may be produced by meiosis. For example, a single human genotype probably carries in the region of 100 000 pairs of genes. If only twenty of those pairs showed differences, one would expect over a million different genotypes in the gametes due to assortment and recombination. In fact, it is likely that many more than twenty pairs of human genes show dissimilarities, so that in an average ejaculate of 200 million sperm it would not be expected that any two of them would share an identical genotype. The same variation will of course apply to egg genotypes, so the probability of any two children of the same parents inheriting the same genotype are quite negligible. Indeed, it is highly improbable that any two human beings who have ever lived will have had the same genotype (except where the zygote has split into two *after* fertilization, giving identical twins).

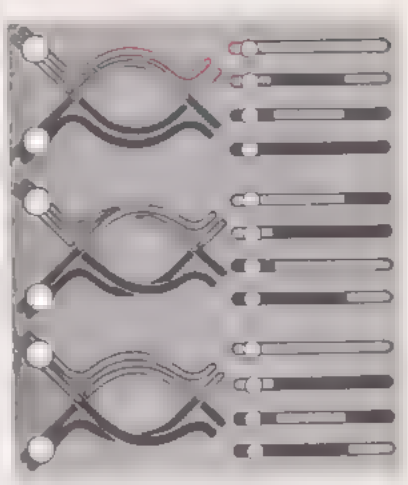


Figure 10 *Diagrammatic representation of various combinations of cross overs between different chromatids.*

The effects of mutation in producing entirely new genes must be remembered too, as these will also be undergoing recombination. Probably 20 per cent of the people around you contain some mutation which arose for the first time in either their mother or their father—and this figure excludes dominant lethal mutations, as you will not have seen these people!

There are other factors giving rise to genetic variation. One of these is the production of individuals with more than the normal set of chromosomes. These individuals are called *polyploids*, and though they seldom survive if they are animals, many important crop plants are in fact giant polyploid versions of a normal ancestor. You will find more about these in black-page Appendix 3.

polyploid

19.3 Genotype and Phenotype

These terms were defined in Unit 17. The variations we have been discussing above are variations in the genotype of the organisms, i.e. the genetic composition of the individuals. These variations will show as changes of some kind in the phenotype, unless they involve wholly recessive genes.

In instances such as the gene for 'vestigial wing' in *Drosophila* the animal either shows the abnormality or it does not. But in other cases a single recessive gene may in fact 'show through', so to speak, even if only to a very small extent. One example is the recessive gene for Haemoglobin S, mentioned in section 19.2.1 above. As you will see in 19.6.4, it is possible to observe the effect in the heterozygote, although only under certain conditions. Thus it would be rash to assume that a single recessive gene never has an effect of evolutionary importance on the phenotype.

During an individual's lifetime, phenotypic changes will occur continuously, an obvious example being growth, maturation and ageing, but changes to the genotype will be small (random mutation) or absent. Furthermore, changes in the genotype will not be heritable if they occur in any of the great majority of the organism's cells – its body cells – but only if they happen to occur in sperm or egg mother-cells in the gonads. Thus, within the life of an individual organism, we expect radical changes to occur in the phenotype, but probably not in the genotype. Nevertheless, the nature of the genotype may profoundly affect not only the original structure and function of the phenotype but also the phenotypic changes which occur.

use affects phenotype

For example, it is a matter of common experience that use (= 'training') is profoundly important in the development of the body for various forms of athletics. For many forms of sport, one of the limiting factors on performance is the efficiency of the circulation in getting oxygen to the muscles (see Unit 18) and in removing lactic acid as it accumulates. A great deal of the difference in stamina between, let us say, an athlete and the authors is, in fact, merely a reflection of the relative capacities of our circulatory systems.

Use not only increases the size of the appropriate muscles, but also develops the blood vessels supplying them. It also greatly increases the amount of blood circulated per minute, sometimes the crucial factor. This is achieved, rather surprisingly perhaps, by increasing the capacity of each heartbeat – i.e. the blood pumped per beat – at all times. The athlete and the authors, when at rest, require about the same amount of blood to be pumped round their bodies, say 5 litres per minute. This will be achieved by the heart beating about 75 times per minute in the authors, but only 60 times per minute in the athlete. In a fierce race, the more blood that can be got to the muscles the better. The authors' heart rates will rapidly rise to 180 per minute, at which rate the amount of blood pumped per beat is falling rapidly, as the heart does not have time between beats to relax and fill properly. Thus this rate, perhaps the maximum, will be reflected in a maximum output of about 25 litres per minute. At this output, however, the athlete's heart is ticking over at a mere 100–120 beats per minute and when pushed harder, it can give an output of 35 litres per minute.

Training would improve the performance of the authors' hearts to give a larger, slower beat, but would never enable them to compete with an athlete. The evidence is that to be able to reach the really high output

figure, it is necessary to start with a slow strong beat. The characteristics of the heart will initially be determined by the genotype, and this will affect what changes can be induced in the phenotype by training. The same training will not therefore produce equally good results on two genotypes. Whereas the athlete may be able to pass such potential on to his children, it is unlikely that the authors will to theirs.

potential determined by the genotype

[illegible] has been a matter for much thought over the years. The Danish botanist Johannsen started an experiment in 1909 in an attempt to distinguish which variations in a variety of garden beans were due to genetic differences and which were phenotypic. The variation he was measuring was in the size and weight of the bean seed itself.

He used a commercial 'variety', which in reality consisted of a number of different genotypes. There was a wide range in the sizes of the beans, and he separated out the smallest and the largest ones. He grew them under similar conditions, and when the plants flowered he self-fertilized them. (That is to say he fertilized the female part of a flower, the *ovules*, with pollen from other flowers on the *same* plant.) This ensured that no new genotype was introduced, only that already present in the original seed from which the plant was grown.

We may call the plants grown from the small seeds group A, those from the large seeds, group B. After each plant had been self-fertilized, the beans grew and ripened in the normal way. He then collected them and measured them.

Would you expect that the average size of the beans produced by the plants of group A were:

(a) the same as the average size of those produced by group B (large seeds)?

(b) smaller than those from group B?

See Answer 3, p. 76.

There was a considerable size range within the progeny of both groups. Johannsen then took the largest and the smallest beans from *within* each group, grew them, self-fertilized the flowers and collected the beans.

He found that the average size of the beans grown from the smallest beans of the group A line was the same as the average size of those grown from the largest of the same group. Thus within the group his selection was not effective.

Why not?

See Answer 4, p. 76.

Thus group A was 'breeding true' for smaller beans, subject to the normal variations expected from the effects of the environment and experimental errors.

The same applied to group B.

Would you expect that the average size of the group B line remained greater than that of the group A line?

See Answer 5, p. 77.

Many other instances of the effects of the environment on phenotypes spring to mind. For example, a young couple may spend their summers on the Costa Brava and their winters skiing—and be elegantly suntanned all the year round. However, they may eventually have children and cannot

afford to go on holiday. Not only will they lose their tan – but their baby will be born without a trace of it.

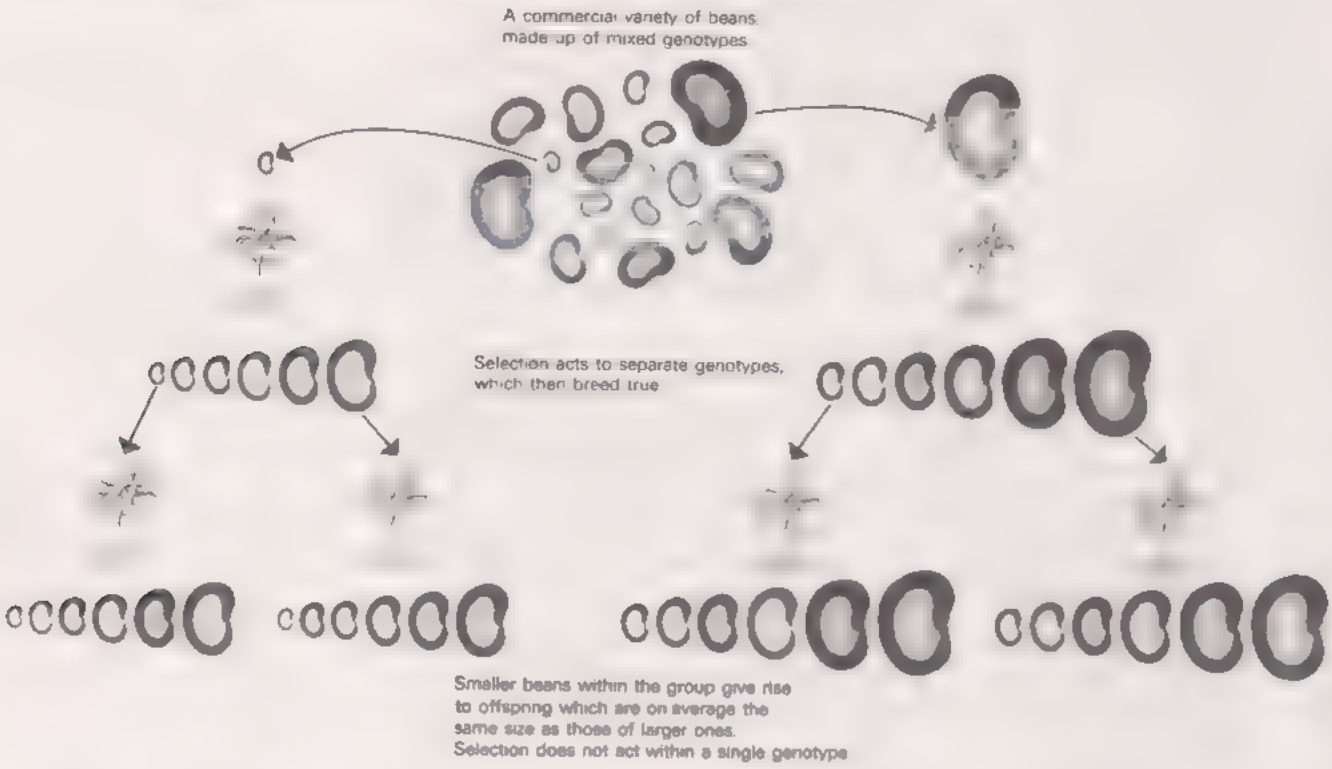


Figure 11 *Diagram summarizing Johannsen's experiment.*

Obviously, the differences in size of the beans possessing a single genotype or in a 'pure line' were caused by variations in the availability of such things as light, dissolved salts, and perhaps the physical nature of the soil encountered by the roots of different plants. The position of the bean in the pod could also have made a difference.

Various species of the garden and pot plant, Hydrangea, show colour variations of their flowers. Thus it is possible to cultivate generations of Hydrangeas with blue flowers, only to discover that if transferred to a soil deficient in iron they become a pale pink. If you want to turn your pink Hydrangea blue, just water it with iron salts; but you will also have to water its progeny, as the change is not heritable.

We have so far considered the effects of the genotype on the phenotype at a molecular level (one gene = one polypeptide—in Unit 17), and also a little of the effects of the environment on the phenotype. What of the effects of the phenotype and the environment on the genotype? Traditionally, the environment was assumed to affect what we would now call the genotype through the phenotype in a very direct manner. Thus the environment, in the form of the crocodile, pulled out the nose of the Elephant's child (the phenotype) and thereafter (→ the genotype) elephants had long noses.

Can you in fact produce a plausible explanation of how this could come about? Many people have, including Charles Darwin, but we will deal with this aspect later (section 19.5). Modern evidence does not support the idea that changes in the phenotype can act on the genes to produce a corresponding change, i.e. one that could imprint that change on the genotype. This must be clear from what has been said both in this Unit and Unit 17. But this is not to say that the phenotype has *no* effect on the genotype. It does, after all, provide the environment for the genes, and thus may affect them. For example, you will see from black-page Appendix I that, if the oxygen level in the tissues drops, mutations of the genotype due to certain causes are inhibited. On the other hand, if the phenotype *Drosophila* is made to live with a higher body temperature, the mutation rate rises.

effect of the environment on the genotype

The external environment in which the whole phenotype lives, also affects the genotype both directly (radiation causing mutation) and indirectly, through the fate of the phenotypic expressions of the genes, that is the individual organisms.

19.3.1 Summary of sections 19.2 and 19.3

1 Although we have seen from Unit 17 that cell multiplication (mitosis) involves a basically conservative mechanism, variation does occur. It occurs by mutation, which is a sudden change in the arrangement of the nucleotides in the DNA of the chromosomes. This results in changes in the polypeptides determined by the structure of the DNA, and so in detectable changes in the cell or organism. Mutations appear to be essentially random, and may occur to any gene at any time, though some genes appear more susceptible than others. Because of the complexity of living organisms, mutations are more likely to be deleterious than beneficial, though when they are recessive they may not affect the phenotype for many generations.

2 Variation in the genotypes of organisms that reproduce sexually will arise because:

(a) each is an equal mixture of material from two parents;

(b) the gametes carrying the material from the parents are produced by meiosis. Meiosis itself involves the random assortment of whole chromosomes as well as genetic exchange between parts of them. Thus almost limitless recombinations of the existing genes may take place;

(c) mutations occur in the cells giving rise to the gametes, as in any other cell.

3 All these variations are heritable, but there will also be phenotypic variations which are not. It may be important to distinguish between them.

Section 4

19.4 Populations

Until now we have considered genetic variation mainly as it may arise in individuals, by mutation, assortment and recombination. So far as the reality of genetic change within any kind of sexually reproducing organism is concerned, certainly from an evolutionary point of view, we have really only been considering models—or at least prototypes. Most organisms do not live as isolated individuals, and even if they did genetic change in such individuals would be hard to locate. But neither do organisms really live together as a species (a term we will examine later) or 'kind'—although we tend to group them as if they did. For example, the genotype of one lion is of little significance to the lion species as a whole, but to try to consider change in the genotype of the whole species becomes impractical, considering that lions may live thousands of miles apart, separated by rivers, mountains and deserts, with no direct communication between them.

If we are to consider genetic change in relation to possible evolutionary mechanisms, with what unit are we involved? Essentially it is a geographical one, though the geography may not always be obvious. It is that group of a particular kind of organism the members of which encounter one another sufficiently often and in such a manner as to result in their interbreeding regularly. Such a group is termed an interbreeding population, or just a *population* for short, and ideally every member of it has an equal chance of meeting and mating with every other appropriate member, but very much less chance of mating with a member of another population.

population

This ideal population may be achieved if the group is confined in a relatively small space with definite boundaries – say rabbits on a small island. More often than not, however, it is more a question of degree. For example, there is almost continuous coverage of the British Isles by the Brown Rat. Even so, we do not normally think of them as providing a single freely interbreeding population, though if we viewed them in terms of the evolution of the whole species over some millions of years we might. But a rat from Glasgow will not often requite its love with one from Tunbridge Wells. For purposes of detailed study by members of the Open University, it would be much more usual to consider the rat population of the village of Milton Keynes and a one-mile radius around it, bearing in mind the possible influences on it of the Bletchley rats, four miles away. All the same, a longer-term study, say over ten years, would require allowance to be made for more mixing and movement, and the net might have to be cast wider. You will meet such an example in section 19.6.1, in the spread of resistance to the poison Warfarin among the rats of Western Britain.

So the boundaries of these population units are often arbitrarily drawn depending on the time scale and the size and mobility of the individual organisms involved, and on where one particular type of environment may be said to end and another begin. Nevertheless, these populations do seem to form the units of evolution and they have a real genetic significance as you will see in section 19.6. Of course, any such population may really only exist for a limited period in time before its edges become blurred.

The main difference between the colloquial sense of the word 'population' and the genetic one is the clear implication in the latter that the members interbreed freely and randomly. Thus two ants' nests close together in a garden would not form a single population from a geneticist's point of view, because they normally breed only within their own colony (and then only a select few are involved). To the average householder, however, it would seem fair to say that his garden had a large population of ants.

Thus a population in the ordinary sense of the word is simply a geographic entity, and can easily be rather larger than the geneticist's one. For example, even today one could not truthfully say that the 'population of London' was a freely interbreeding unit. It is not really true to say that your chances of meeting and marrying the boss's daughter from Primrose Hill are as good as those of marrying your next door neighbour's in Camden Town. It most certainly is not so if either you, or she, is a coloured Moslem from Southall. Thus genetically speaking London is a number of separate populations—perhaps to the detriment of all.

19.5 Evolutionary Theories

In the introduction, we gave our reasons for making the assumption that species had developed by some process of evolution, rather than by any process of special creation. It is clear that such evolution cannot take place unless change can occur in the genotypes. It is this change and some of the causes of it that we have been examining so far. But, unless there is also a great deal of genetic stability or continuity (Unit 17), it is equally impossible to visualize any evolutionary process. If organisms came out quite differently at each generation, no character could be said to be heritable and any idea of a progression to a new or different 'kind', which is what is implied by the idea of evolution, would be meaningless.

So what we are considering is a fairly balanced system, basically stable but with an ever-present element of variation; the units of this system, and those of evolution itself, are the interbreeding populations discussed above. No case for any evolutionary mechanism—or for that matter for its occurrence at all—can be convincing unless it can be shown that it can account for a lasting and directional change in such a population. Indeed, it is far more convincing if the postulated mechanism does not merely accord with the changes, but can be shown to produce them, though clearly even this is not proof positive of its universal applicability.

You must remember that the arguments over the nature and causes of evolution have been taking place for many years, but, until recently, without the genetic information you now possess. The benefits of hindsight are considerable when looking back on the early theories from the vantage point of the 1970s.

Even so, it is quite surprising that three or four hundred years ago, when almost no one would have even considered the heresy that one type of animal could have evolved from another, there was general acceptance of the idea of the inheritance of characters acquired during the life of the phenotype. Ancient folk tales are to be found from all over the world on the general theme of the Kipling story of the Elephant's child, mentioned above. For example, hares are supposed to have split lips as a consequence of the uncontrolled laughter of a bygone Chinese hare.

The implication that generations of earlier hares had intact lips—and that if lips could change, so could ears, fur, size or anything else—seems to have worried no one.

However, by the end of the eighteenth century, a good many thinkers had come to the conclusion that there *had* been organic evolution, though it had not been formulated as a scientific theory, nor had any convincing mechanism been put forward to account for it. The idea was not acceptable to the public at large and was in direct conflict with the teachings of the Christian Church. However, the power of the Church to control philosophy and discussion was on the wane, and it is probably no accident that it was at this point in history that an evolutionary interpretation came to be put on facts which had been under consideration by natural philosophers since Aristotle's time (384–322 BC). Scientists, like others, tend to operate within the social climate of their time. Belief in the immutability of a way of life and the idea that nature has already been explained, is a considerable encouragement to the belief that species are also immutable. Buffon (1707–1778) produced what was probably the first clear statement of belief in organic evolution, and he did so working in Paris in the climate

which led to the French Revolution, a time when many sacred cows were slain. (See *The Roots of Present-Day Science*.)

Erasmus Darwin (1731–1802), grandfather of Charles Darwin, wrote as an evolutionist who believed that all life—including, by implication, human life—originated from a common source. Whereas Buffon was clear that the mechanism was the inheritance of acquired characteristics (see below), Erasmus Darwin avoided coming to grips with this part of the problem. Lamarck (1774–1829) was a brilliant biologist with a very wide knowledge of both plants and animals. He was much influenced by Buffon's thinking—indeed he tutored Buffon's son—and he formulated the first really clear and comprehensive evolutionary theory.

inheritance of acquired characteristics

Like Buffon, he believed firmly that the mechanism was the inheritance of acquired characters, so that today as a 'doctrine' it still bears his name —Lamarckism. The problem encountered by all these men, and some of those who followed them, was the need to explain how variation could occur and the variants be sustained and perpetuated. Common sense suggested that offspring were a blend of their parents, and the blending was usually held to be one of bloods. However, such a system would produce increasing uniformity, and a particular character would become progressively diluted at each generation. Thus, the idea that phenotypic changes are passed on (in the blood) was not only plausible, it also provided an explanation of how new characters could be sustained and improved. If, for example, both parent giraffes somewhat increase the length of their necks reaching up into the leaf canopy of trees, their offspring will have longer necks. The parents are sharing the same environment, so *both* their necks are affected—reducing dilution. The offspring are again confronted with the same environment, so the effect is progressive. Lamarck was quite clear that it was the action of the environment on the organisms which produced the change, rather than any vague accident or unspecified design of the Creator. Many of his views on the interaction of the organism and the environment, and on the nature of the evolutionary process generally, were close to those held by a majority today. His importance in the development of modern evolutionary theory is often underestimated. He did not in fact adopt the very naïve views sometimes called Lamarckist. He quite specifically rejected the idea that crude direct effects of the environment on an organism would be transmitted in any way to its offspring. Thus, he would not have expected that cutting the tails off the adults of generations of mice would result in the development of a tail-less breed of mouse. Yet this experiment has been done at length in an attempt to disprove 'Lamarckism'. In Lamarck's view, the heritable element was the change produced in the phenotype by the phenotype itself, by use or disuse. The whale's flippers or the kangaroo's forelimbs could be considered classic examples of the results of disuse in this context.

One of his main opponents in this evolutionary thinking was Cuvier, whose theory of catastrophism was mentioned in the introduction. But Cuvier's views were largely demolished by the work of the British geologist Sir Charles Lyell (1797–1875) (see Unit 26). He provided convincing evidence that geological evolution was a continuous, and continuing, process rather than a series of Creator-induced catastrophes.

Thus, during the latter part of the eighteenth century and the early part of the nineteenth, scientific thinking was largely along evolutionary lines. However, the only mechanism which had been advanced and supported in any detail to account for biological evolution was that of the inheritance of acquired characters, and their fixation in the species by the action of the environment.

This remained substantially true until 1858. That year saw the joint publication of a paper, based on two separate pieces of work, by Charles

Darwin (1809–1882) grandson of Erasmus, and Alfred Russell Wallace (1823–1913). In this paper, which was read to the Linnean Society of London, the authors suggested that the main factor producing evolutionary change was what they called *Natural Selection*.

natural selection

Their argument, amplified a year later by Darwin in *The Origin of Species*, was essentially this: organisms tend to produce more offspring than their environment will support, therefore a large number will perish before completing their reproductive lives. Those whose phenotypes are better suited to their immediate environment will, in the long run, have a greater chance of being among the survivors than the less well suited. This in turn will mean that over a period of time the better suited phenotypes will predominate in the population.

The ingredients of this process are (a) *overproduction* of the organisms in terms of what its environment will support thus leading to (b) *competition* among (c) heritable *variations* of the phenotype.

overproduction
competition
variation

There is no doubt that these ingredients are normally present within any natural environment. Overproduction is quite evident. A plaice lays half a million eggs at a single spawning, a cod several million and an oyster over 100 million. A pair of rats may produce an average of ten young in a litter, three or four times a year. The offspring themselves will mature in three or four months, with the result that a pair of rats in an ideal environment could be one of five hundred pairs within a year.

Bacteria dividing every thirty minutes could soon engulf the Earth's surface, and if every one of the 700 000 000 000 spores of the puffball fungus were successful, one such could in theory give rise to a mass of puffballs greater than the Earth in eighteen months or so. Common experience shows that all the organisms we meet have a much greater reproductive potential than they are likely to be able to achieve for long. Man is in the interesting position of being able, so far, to 'bend' the environment continuously to keep up with almost unrestrained population growth, though even this growth is less than he is theoretically capable of.

Given the first ingredient, the second must almost inevitably follow Taken at its simplest, if there are 100 animals born into an environment which only provides enough food for twenty-five to grow and mature, there will be great pressure on all the individuals and they will be directly in competition with one another. As the food supply becomes heavily taxed, they may all become undernourished. At an individual level it may be pure chance which one succumbs and which survives. One which has been more successful in feeding itself and is thus more active and stronger may nevertheless be caught by a predator, but all the same its chances of survival are better; in the long run the more successful food gatherers would be expected to survive to maturity more commonly than the less successful. Clearly the third ingredient is involved at this point. If all the 100 individuals are identical, it will only be pure chance that determines the survivors. If they are genetically identical but show phenotypic differences, any advantage conferred upon an individual by such a difference would only be passed on to its progeny if there is in fact some direct effect of phenotypic change on the constitution of the germ cell, which most geneticists would deny. In fact, the animal would be in the position of Johannsen's beans belonging to a pure line—even when there was 100 per cent selection of the larger beans within the line, the beans produced by these plants were no bigger on average (p. 25). This experiment, together with what you have seen of modern genetical theory and research, make it almost certain that Darwin's 'natural selection' will only operate to produce lasting change in a population if there is *genetic* variation within that population.

competition from overproduction

When Darwin and Wallace were formulating their theories, however, they did not know this. Although the first steps in genetics were taken by an

Austrian monk named Gregor Mendel and published in 1865, during the time that Darwin was actively improving and elaborating his theory, he never had the benefit of them. Mendel published his results in a single paper in the Brno Natural History magazine, not a widely read journal, and indeed they remained unnoticed until the basic 'rules' of genetics were rediscovered, as was the paper, in 1900, some time after Darwin's death. Had Darwin known of them, it probably would have made a great difference to the development of his theory. He always felt that the weakest point of his argument lay in the absence of an explanation of how variation was transmitted. Not having any of your knowledge of genetics, he never believed the observable mutations in domestic animals ('sports', as they were called) to be a major factor in providing the variation on which natural selection could work. He assumed that such changes were bound to become more and more diluted at each generation, in a form of 'blending inheritance'.

Darwin is commonly held to have produced 'the' theory of evolution, and to have accounted for it by the idea of the 'survival of the fittest'. But neither of these statements is really true. As we said above, the idea of biological evolution was a topic of much discussion among scientists and natural philosophers during the latter half of the preceding century. Darwin's great contribution was to have suggested, in his hypothesis of Natural Selection, a practical and easily understandable means by which evolution could have taken place.

What Darwin did was to put the idea of organic evolution into the framework of a real scientific theory and to present an enormous amount of careful observation and reasoned argument to support it. He himself accepted evolution as fact at the start of his work; his main theme was to account for adaptation and to explain evolution in terms of it. The idea that natural selection was the agent by which the environment affected species (and indeed that this could lead to entirely new species) was entirely original and of the greatest importance. It is essentially the view held by most biologists today. But Darwin believed that natural selection was merely the most important of four major factors. The next most important was the inherited effects of use and disuse (i.e. 'Lamarckism'); then came the inherited effects of the direct action of the environment on the phenotype (an occurrence Lamarck did not accept, but one adopted by his followers or 'neo-Lamarckists'); and finally the occurrence of 'sports' or mutants. The condensation of the idea of natural selection into the phrase 'survival of the fittest' was not Darwin's, but an enthusiastic follower's, Herbert Spencer. It is rather misleading, suggesting that the process produces the very best in some absolute sense, rather than simply tending to eliminate more of those members of a population less well adapted to a particular environment. However, it was a phrase that caught the imagination, particularly of those interested in its application to human society and ethics.

Thus Lamarck and Darwin were really not so very far apart in their thinking, though this is not to belittle the extent and accuracy of Darwin's work or the overwhelming importance of the idea of natural selection. It is rather ironic therefore to find that bitter controversy raged throughout the end of the nineteenth and the beginning of the twentieth century between 'neo-Lamarckists', who postulated direct environmental effects that Lamarck did not believe in, and the 'neo-Darwinists', who interpreted natural selection in ways Darwin would probably have laughed at.

19.5.1 Can these theories be verified?

It is, of course, nearly impossible to *disprove* either theory, or for that matter to *prove* that any such theory has accounted for all evolution; it is not enough, even today, to say that our knowledge of genetics shows that Lamarck was wrong. It makes it likely that he was wrong because it is hard to think of a Lamarckian mechanism compatible with what we know (unless, of course, *you* did on page 26). But what we *can* do is to make predictions according to either theory, and then test the predictions. This is not very easy to do for several reasons, one of which is the time-scale involved.

You have, however, already met one example where the generation time of the organism is as little as thirty minutes and evolutionary processes which would take thousands of years in a mammal could perhaps be telescoped into months. This example is *E. coli*, the bacterium cited when we considered mutations arising in populations of it in culture (p. 11). These mutations conferred a resistance to the antibiotic streptomycin, in one case, and to attack the T4 phage, in another. Exposure of a population of *E. coli* to these agents represents a fundamental change in its environment, in the first case by subjecting it to a massive attack by a parasite not previously present in its environment, and in the second by saturating the environment with a chemical that upsets the replication process in the normal bacterium. Both of these are rather acute examples of the sort of change which could be encountered by any natural population.

selection or induction

Not all the individuals were killed; in both cases some survived and formed new colonies, the genetic constitutions of which were different from those in the original population. In the case of phage resistance, the experimental technique used is as follows. The bacteria are grown in a liquid medium, let us say in ten tubes, each tube being seeded with one or a small number from a single clone. These are then incubated until the bacteria have multiplied to a density of about 5×10^9 per cm^3. Whilst the bacteria are being incubated, ten Petri dishes are prepared with a nutrient jelly containing the bacteriophage in very large numbers, much in excess of those of the bacteria. The bacterial cultures are then poured onto the jelly containing the phage and the dishes are incubated. Sensitive bacteria are destroyed in a few minutes. Any resistant ones will grow into visible colonies within twelve to sixteen hours.

As we said above, these resistant ones must have a different genotype from the normal population, which is sensitive.

How would you account for this change in Lamarckian or neo-Lamarckian terms?

See Answer 6, p. 77.

How would you account for them in terms of modern 'Darwinian' theory?

See Answer 7, p. 77.

In terms of these two theories, can you make predictions as to what the outcome of the experiment should be, numerically speaking?

See Answer 8, p. 77.

Table 2 gives the results actually obtained from five experiments performed in the manner described above. Each figure represents the number of resistant colonies in each of the ten dishes (in the case of experiment 5 only nine dishes were used).

Table 2 Number of resistant colonies in experiments with *E. coli*

	Experiment No.				
Dish No.	1	2	3	4	5
1	30	6	1	1	10
2	10	5	0	0	18
3	40	10	0	0	125
4	45	8	0	7	10
5	183	24	0	0	14
6	12	13	5	303	27
7	173	165	0	0	3
8	23	15	5	0	17
9	57	6	0	3	17
10	51	10	107	48	—

Do these findings agree with the Lamarckian prediction? Clearly they do not. They do, on the other hand, allow the 'Darwinian' view to be held. The distribution of resistant colonies is in fact what would be expected on statistical grounds from a spontaneous mutation rate between 1.1×10^{-6} and 4.1×10^{-6}.

Thus we have firm evidence of an instance where a major change in a population has been brought about by selection of a genetic character which had already arisen in the population, furthermore, it is an instance where the so-called 'Lamarckist' prediction is not fulfilled. A much simpler and cruder way to test the 'neo-Lamarckian' theory, that is the direct effect of the environment on the genotype, is to perform some operation on many generations of adults, to see if the change begins to appear in the offspring. This poses many problems, not least the question of *when* the phenotypic change is expected to affect the genotype. After all, it is known that in the female mammal most, if not all, the egg cells are present in the ovary even before the female is born, therefore one could argue that the phenotypic change might have to occur before birth. Nevertheless, enthusiastic biologists cut the tails off hundreds of generations of mice without producing the forecast strain of 'manx' mouse. Dog breeders have been docking the ears (though no longer in Britain) and tails of Boxers for a great many years, but the puppies are still born with long ones.

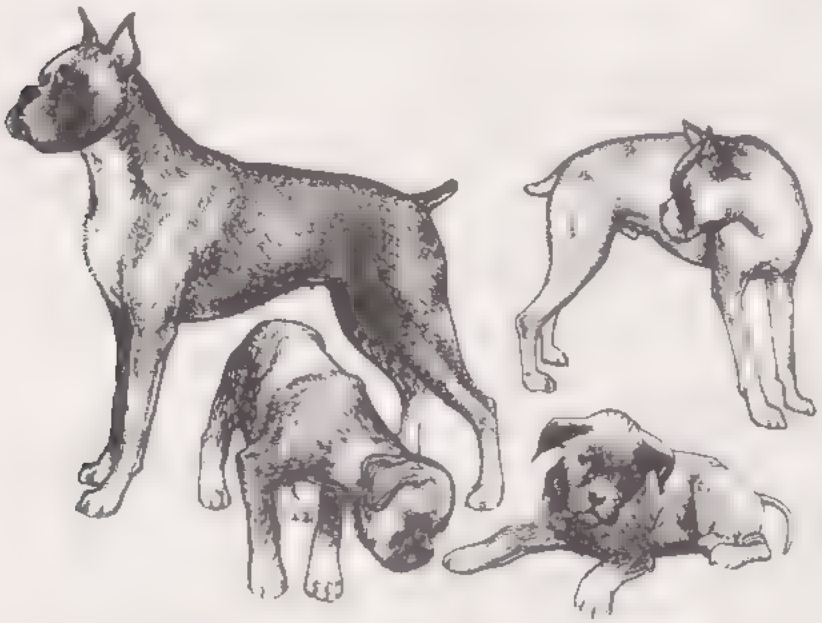
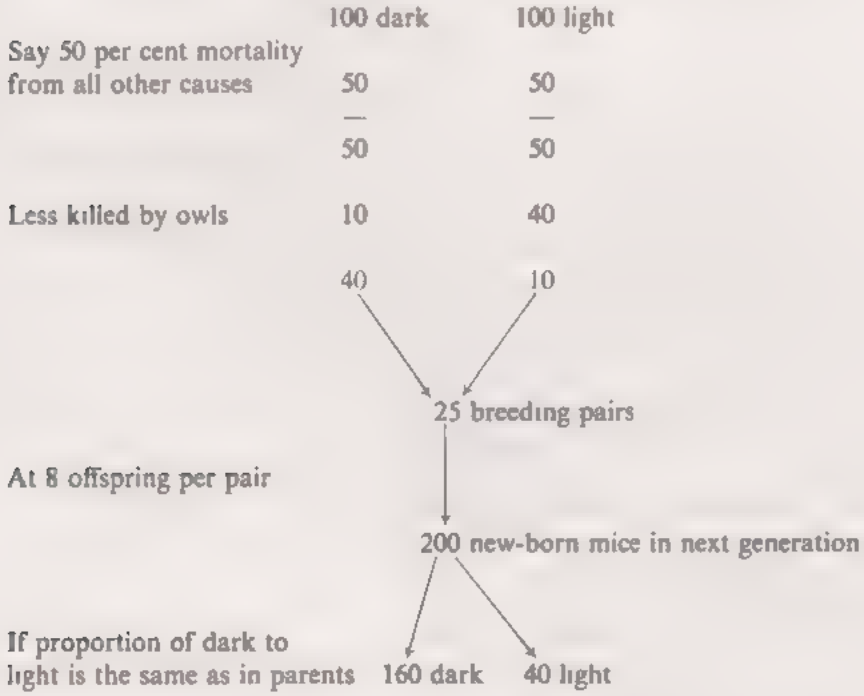


Figure 12 Parent Boxer dogs with docked ears and tails and their normal puppies.

19.6 Micro-Evolution

We have established that the environment produces change in a population by favouring some heritable variations within it more than others. We have said that the favoured organisms are the more 'fit'. Used in the evolutionary sense, '*fitness*' does not mean quite the same as in the athletic sense. Observation may suggest that an animal is better fitted to a particular environment than others of its kind because it is better concealed, or because it is bigger, or because it seems more intelligent. This is really trying to put a value-judgement on a character in a situation which is so complicated as to make it meaningless. The proof of the pudding is in the breeding, which is to say that the only meaningful way to judge fitness is in relative numbers. If a variety within a population is common, it is reasonable to say it is well fitted to its environment. If it appears to be increasing or have grown at the expense of others, it is also reasonable to say it is better suited, or more fit, than they are. All the many factors really boil down to this one end factor, which can usually be measured. Thus evolutionary fitness is really reproductive fitness. An observer may form an opinion as to *why* a variation is more (or less) successful, but the only way he can measure it is in terms of survivors over several generations. We can use an imaginary example of how selection may be observed to determine 'fitness' in the wild. Consider a population of mice, equal in all observable respects (a statement one would not dare make in a real example) except that half are light coloured and half are dark coloured. If the main predators are a pair of owls, higher losses are to be expected among the lighter variations, as they are more conspicuous at night. It is reasonable to assume that each pair of mice will average eight offspring per litter. We can start with a population of 200 new-born mice:

reproductive fitness



Thus the owls will soon make the light variations a rarity. We can put a figure to this 'fitness'. We can say that of the original 100 dark mice, 40 survived to breed and took a part in the production of $40 \times 8 = 320$ offspring. To maintain a stable population where there are equal numbers

of males and females, the average production of offspring must be two per pair. Assuming that this was the only breeding of our original mice, we can say that they averaged 3.2 offspring each rather than 2 or $\frac{3\,2}{2}$, giving them a 'fitness' of 1.6. By the same calculation the fitness of the light mice under these circumstances was 0.4.

A real example is demonstrated in the TV programme of this Unit, when we deal with the case of the mutant form of the Peppered Moth. You will notice there that we do not refer to fitness as a ratio, but we mention simply the 'survival advantage' of one form over another as a percentage. This is because we do not have all the figures of the reproductive sequence given in the imaginary case, we are merely measuring a relative adult survival.

In the case of our imaginary mice, natural selection was producing a minor change in the population; within a few years it would have made light coloured mice a rarity. They would probably not have vanished altogether, for various reasons—new ones might have arisen by mutation, or migrated in from areas where the predators were not owls or where their surroundings were lighter, thus giving them a better chance.

Small but important changes due to selection can be shown to be occurring in real populations, and these small evolutionary steps are often said to be examples of micro-evolution, to distinguish them from major evolutionary steps such as the production of clearly distinct new species, such as we shall see in section 19.7. The change occurring in the population of the Peppered Moth, mentioned above, is an example of micro-evolution.

Many of the processes of adaption occurring as a result of selection pressures are not of merely scientific interest, but of the greatest immediate importance in our everyday lives. One common example of this is *adaptive immunity*, the acquisition by a population of immunity to agents, biological or chemical, which previously were severely damaging.

19.6.1 Adaptive immunity

bacterial resistance to antibiotics

You have seen how strains of *E. coli* resistant to antibiotics can be isolated in the laboratory. The same process, unfortunately, may occur within human communities to much more harmful bacteria (remember the radio programme of Unit 10). *Staphylococcus aureus* is a common bacterium found on the skin, in putrefying matter and elsewhere. If it gets into the tissues it is pathogenic, that is to say, it may multiply faster than the body's defences can destroy it, attacking the tissues and producing poisonous waste products. Depending on where it happens to be, it may be the causative organism of a sore throat, boils or a fatal blood poisoning. This bacterium, in common with other staphylococci, is generally very sensitive to penicillin, even in quite small doses. However, when a dose of penicillin just large enough to 'cure' the disease is used, a number of resistant individuals able to protect themselves by producing a penicillinase, are likely to be isolated—as in the culture of *E. coli*. These will not necessarily succeed in multiplying into large numbers (causing a relapse in the patient), because the normal bodily defence mechanisms may keep them in check. Nevertheless, they will be there in small numbers, perhaps in the discharge of a healing boil or the saliva of a recent victim of a septic throat. Eventually, one such resistant bacterium may infect another victim, and it will be found that the disease does not respond to treatment with the normal dose of penicillin. Usually a very much larger dose will still work, and so it will be used. Resistance to very large doses requires a further mutation, in addition to the first one. But once again the selection

process may take place, this time producing bacteria resistant to very large doses, often as large as it is safe to prescribe. It is in this way that modern medicine produced the 'Hospital Staph', a very tough organism, with the result that the level of post-operative infection—which had fallen very low indeed in the early days of antibiotics—has risen considerably. Usually the causative organism is an antibiotic-resistant one.

The first signs of staphylococcal resistance appeared soon after penicillin became extensively used in hospitals, as Table 3 shows.

Table 3 Incidence of penicillin-resistant infection at a general hospital

Date	*Total patients*	*Patients with penicillin-resistant strains*
Apr–Nov, 1946	99	14
Feb–June, 1947	100	38
Feb–June, 1948	100	59

By 1950, a majority of staphylococcal infections in *all* British general hospitals were penicillin-resistant.

Resistance has subsequently appeared in certain staphylococci to all the major antibiotics, often as a triple resistance, that is the same strain being resistant to penicillin, tetracycline and streptomycin. Fortunately, not all the strains of *S. auraeus* have produced such a dangerous triple immunity.

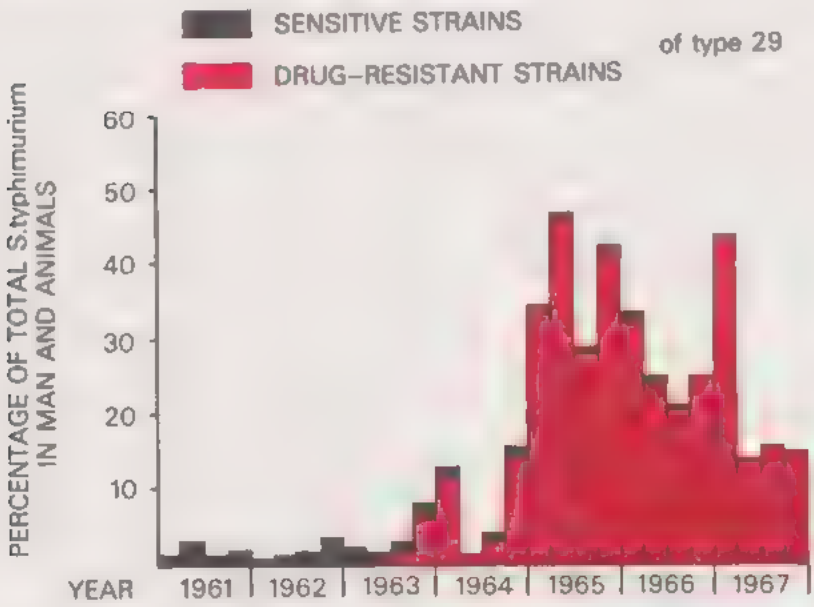


Figure 13 Incidence of drug-resistant strains of the type 29 bacterium causing food poisoning in man and animals, expressed as a percentage of the total number of diagnosed infections.

bacterial resistance to sulphonamides

It is also interesting to note that the much more dangerous bacterium *Streptococcus*, which was producing strains resistant to treatment with sulphonamides† (in the days before antibiotics were used), has fortunately failed to produce a penicillin-resistant strain. There are, however, mutant strains resistant to tetracycline. Similarly, the bacterium causing the venereal disease gonorrhoea produced, as a result of selection, strains resistant to sulphonamides; more recently strains with partial resistance to penicillin and streptomycin have emerged. Figure 13 shows the dramatic rise in infections by resistant strains of *Salmonella typhimurium*, the causative organism of food poisoning.

We can see with the benefit of hindsight that the incidence of this resistance might have been much reduced, or delayed, by not prescribing antibiotics unless really necessary and, more important, using larger doses in the first place so that the chance of there being survivors was reduced. The chances of the double mutation (to give resistance to large doses) having occurred spontaneously without the effects of antibiotic selection are very small. If the first mutation carries no advantage, and remains rare, the chances of the second mutation occurring among those few individuals is very slight. Thus, if we had used, say, 100 rather than 20 mg/litre of streptomycin in our cultures of *E. coli*, we would not have expected any survivors in the Petri dishes.

In modern medicine, therefore, it has to be accepted that any pathogenic bacterium may give rise to strains resistant to almost any antibiotic. This means that the situation is always a dynamic one. New strains evolve; new antibiotics are produced to beat them. Slight changes in the structure of the drug are sometimes sufficient, and by an empirical process of 'molecular roulette' and testing, research departments in the pharmaceutical industry and elsewhere have been able to keep ahead of the adaptive changes in the bacteria. This has been discussed in the radio programme of Unit 10, so you may recall how this has been achieved at the molecular level, particularly by the production of new penicillins.

DDT resistance

DDT resistance in insects

A similar selective process has occurred among several insect species. As early as 1947, reports began to appear of strains of the housefly resistant to the chemical dichloro-diphenyl-trichlorethane (DDT). This chemical is poisonous to most insects in very low concentrations, and its introduction as an insecticide two or three years earlier had marked a real breakthrough in pest control. However, in various areas in countries all over the world, populations of flies soon appeared which were not killed by DDT in the concentrations originally used. As with the bacterial example given above, the dose had to be raised to be effective, but this was only possible within limits, as very large doses of DDT are not safe for either man or domestic animals (indeed, at the present time, it is not considered desirable even in quite small doses). As a result, other chemicals have had to be developed to replace DDT in many parts of the world.

Under laboratory conditions, a significant degree of resistance has been evolved within three generations in the housefly, by a process similar in principle to that used to produce antibiotic-resistant bacteria.

DDT-resistant strains have also appeared in many other insects, including some mosquitoes, body lice, bedbugs and cockroaches. Some insects have even evolved strains resistant to poisons such as prussic acid and lead arsenate.

Resistance in mammals

It is not only such prolific organisms as bacteria and insects that are able to adapt fast enough to embarrass us in our attempts to exterminate them. Recent instances involving mammalian pests provide similar examples.

For some years the Brown Rat (*Rattus norvegicus*) has been very effectively controlled by a poison known as Warfarin. This contains a substance called di-coumarol, which, if it is eaten regularly, interferes with the clotting processes of the blood. If this happens the rats become, in effect, haemophiliacs†. They gradually weaken, and finally die from minor bites or bruises, or when having litters. They do not associate their condition (and that of those around them) with the food, and continue to eat it, which is why the poison is so useful. (Where acute poisons such as phos-

phorous are used, the survivors become wary, and stop eating the bait and so cannot be destroyed in the same way.)

Warfarin resistance in rats

In 1960, strains of rats appeared in Shropshire and Worcestershire which were unaffected by di-coumarol and indeed were flourishing on the poisoned bait. The incidence of the resistant individuals in the populations of these counties and of parts of Wales had risen to about 50 per cent in 1970, presumably because of the intense selection pressure provided by the widespread use of Warfarin. An interesting aspect of this resistance is that it appears to be due to a single mutant gene, and individuals which are homozygous for this gene are in some other respects weaker than normal (Warfarin-sensitive) rats. We will return to this aspect in section 19.4.4.

The physiological details of this resistance are poorly known, but, in spite of intensive efforts by the authorities, this strain (or strains) is now spreading fast towards the industrial Midlands. Attempts to contain the spread by surrounding the areas with a belt in which old fashioned methods of extermination are intensively applied have not been successful. If the new strains reach the large towns, the rat population, already more numerous than the human one, is likely to rise dramatically.

A second example is provided by the rabbit. This animal has been something of a pest of arable crops and grassland in Europe (Unit 20) for two hundred years, though offsetting its nuisance value somewhat by providing a source of food and felt hats. It became a far more serious pest after its introduction into Australia, where the tremendous efficiency of its reproductive system compared with that of the native pouched mammals (Unit 21) enabled it to multiply almost unchecked.

myxomatosis

Attempts were made both in Australia and Europe to control it by the introduction of a virulent virus disease, myxomatosis, which is endemic among a species of rabbits in South America. This was very nearly successful; in many areas well over 90 per cent of the rabbit population was destroyed. It looked for a time as if the level would fall so low that the animal would indeed become extinct over wide areas. However, in Britain at least, it became apparent that the disease was not hitting all the population, because a small percentage of it was, for some reason, living above ground in nests, rather as hares do. Rabbits of course normally live in large crowded warrens underground and, as the transmitting agent of the disease in Britain is the rabbit flea, the disease spread fast. It became, however, a very effective selective agent favouring those individuals who lived relatively solitary lives above ground—they only rarely caught each other's fleas, and thus each other's myxomatosis. (Under normal conditions they were probably less successful, this open-air habit leaving them more at risk from predators.)

This resulted in something of a rally in the rabbit population by individuals who were not necessarily physiologically resistant, but whose behaviour protected them to a considerable degree.

resistance to myxomatosis

In the Australian rabbit population however, it appears that a genetically resistant strain emerged after the initial epidemics. The investigation described below (Table 4) was undertaken on rabbits from the Lake Orana district. Precautions were taken to ensure that none of the rabbits tested could have acquired immunity by having caught and survived the disease (in the same way as you may have an immunity to measles as a result of having had it in childhood), or by receiving a temporary 'inoculation' against it from their mother's bloodstream before they were born.

In order to make sure that the change was in the rabbits and not in the virus (see below), the virus used was from the original outbreak. It was stored, and each year some of it was tested on laboratory rabbits to confirm that its virulence had not declined.

Table 4 Investigation showing the emergence of a strain of rabbits resistant to myxomatosis

Test rabbits, taken from the wild population		*Numbers of epidemics of myxomatosis previously suffered by the population*	*Symptoms*		
			Fatal %	*Moderate* %	*Mild* %
1	Prior to arrival of myxomatosis	0	93	5	2
2	in 1953	2	95	5	0
3	in 1954	3	93	5	2
4	in 1955	4	61	26	13
5	in 1956	5	75	14	11
6	in 1958	7	54	16	30

In Britain, although the mortality declined in the later epidemics, there seems to be no evidence that this was due to a genetic change in the rabbits. Apparently a mutation occurred in the virus which made it much less often lethal to the rabbit. This quite frequently happens with disease-producing organisms, and because such a mutation is advantageous to the organism the mutant form is likely to become the predominant strain. (Clearly a parasite which kills its host too soon commits suicide, whereas if a balance can be struck it can remain in the population indefinitely.) If its victims survive an attack they will acquire a degree of immunity to it; and if this happens to many individuals, even an epidemic of the original strain will probably not kill them, as they will have at least partial immunity to this as well.

mutation of myxomatosis virus

Would the immunity acquired by a survivor be passed on to its young?

No, it is a phenotypic change only.

(Temporary immunity may be passed on by a mother to her young from her blood system, before they are born, or through her milk, but this immunity is not only transient, it is really only a temporary 'inoculation' by the mother—nothing to do with the bodily capabilities of the young themselves.)

These examples illustrate clearly two general points, the first being the fundamental one, that selection (in these cases as applied by man) will act on variation to produce a suitably adapted population. The second follows by implication: this adaptation means it is foolish to expect a 'wonder drug' to solve a pest or disease problem indefinitely. The situation is a dynamic one and unless this is appreciated from the outset, only temporary relief will be obtained.

19.6.2 Adaptation to exploit new food sources

If new food sources are introduced into an environment, any organism able to exploit them will be at an advantage. Sometimes this ability requires only a change in behaviour, but in other cases profound physiological changes are needed.

Many man-made substances, for example plastics, are potentially rich sources of energy; but they are new molecules so far as living systems are concerned, and so would require new and specialized enzymes to digest and metabolize them. They are also often long, condensed molecules, and these are anyway difficult to attack—few organisms can digest either hair (keratin) or cellulose, both of which have been around for a long time.

It is probably true that the more complex an organism's nutritional system has become, the less likely it is to be able to evolve completely new

digestive and metabolic pathways. This, coupled with the very rapid generation time of many simpler organisms, means that we would expect to see any evidence of the ability to exploit these molecules arise first of all among so called 'lower' organisms. In fact, this appears to be the case. Whereas both rats and human babies have chewed plastics since they have been in general use, so far only simple moulds seem to have been able to use them as food. Poly-vinyl-chloride (PVC) has been considered absolutely immune to rot of any kind; but recently thousands of pounds worth of damage has been done to the PVC insulation of stored army electronic equipment by a mutant mould. Another surprise for the Armed Services came when the shock-absorbent undercarriage of some naval aircraft suddenly went rigid. This proved to be caused by millions of micro-organisms clogging valves in the hydraulic system—they had been feeding on the hydraulic fluid, which is made up basically of hydrocarbons.

There are now mutant moulds which will attack the plastic in household emulsion paints, so that fungicides must be added to prevent your bathroom walls becoming a culture medium.

The examples are many, but the message is the same. Whenever a mutation gives rise to an enzyme which will allow a new molecule to be used as a food source, the advantage that this confers on the possessor may well lead to its multiplication throughout the environment. This can be checked by the addition of poisons, but there is always the likelihood that resistance to these will arise. Again we can see that the situation is a dynamic one.

19.6.3 Deliberate selection

Possibly the most important examples of the selection of variants, as far as human beings are concerned, are found in the field of plant and animal breeding. Nowhere near the present world population of human beings could be fed had not 'artificial' (i.e. human) selection produced new species and strains of crops and domestic animals. For example, the yields that could be obtained from wild cereals, if these were planted in the acreage today available to man, would satisfy less than half his present energy requirements.

selection of crops and domestic animals

The prognosis of the food supply and demand situation is somewhat depressing; even so, it leans heavily on the assumption that selective breeding will continue to increase productivity at least as rapidly as it has done over the last fifteen years or so. Thus, although the most familiar examples of selective breeding may be found among dogs, or in Shetland ponies, shire-horses and racehorses, the most important examples are to be found in such instances as the transformation of wild ancestral maize and rice into the modern high protein varieties, and the production of wheats resistant to attack by rust fungus.

This is not to say that the selective breeding of animals is without importance, particularly to those of us in countries where animal protein forms a large part of our diet.

A recent survey comparing poultry production in the 1960s to that in the 1930s showed that a modern bird not only grew much more rapidly, but that it required only half the food per pound gain in weight compared to the 1930 bird. A new breed of pig has just been produced which reaches bacon weight in 150 days instead of the current norm of 185 days; and on 20 per cent less food. The genetics of this sort of change are immensely complicated, as many different genes are involved, but the approach is much the same as in the development of new crops.

Until such time as genetic material can actually be manipulated to give man what he needs, the only available method is the careful and informed

selection of mutants or recombinants. The crude mutation rate can be accelerated, but this is seldom helpful in practice. However, if the relevant genes can be located on the chromosomes, the process of recombination can be shortened. Many of the techniques employed are very sophisticated, but the principles involved are those you are already familiar with

19.6.4 Apparently anomalous selection

In order to predict the likely outcome of a particular selective pressure on a population, it is necessary to have a fairly complete knowledge of all the factors involved. This information will be very difficult to obtain for a natural population in the wild. Consequently, the adaptive response observed may appear distinctly unexpected.

sickle-cell anaemia

Just such a situation has been observed during studies of the disease sickle-cell anaemia, described in section 19.2.1. You may recall that this was caused by a mutation which resulted in the incorporation of an incorrect amino acid at one point in the protein chains of the haemoglobin molecule. You may also remember that the mutated gene known as Haemoglobin S (or Hb S) is recessive, thus only those persons having the sickling gene from both parents (i.e. homozygous for the gene) actually suffer from the disease (Fig. 14). These sufferers are gravely disadvantaged, and have only a 20 per cent chance of surviving to maturity as compared with normal babies in their community.

What would you expect to be the fate of a mutated Hb.S gene in a population? Would you anticipate that it would:

(a) spread steadily throughout the population as time went by;

(b) rise to a particular frequency in the gene pool of the population say 20 per cent and then level off and remain constant;

(c) be slowly eliminated as it appears in the phenotype (i.e. when inherited from both parents)?

You would expect it to be eliminated as it became frequent enough to start appearing in both of the homologous chromosomes. The disease is a very harmful one, so that the gene is almost a lethal mutant when homozygous. However, *see the following text.*

It is clear that we would not expect the mutation to be favoured, indeed a harmful mutation of this sort is normally eliminated. This will happen rapidly where it is a dominant one, more slowly where it is recessive, as selection can only act on a trait when it appears in the phenotype. New mutant Hb S genes would obviously arise spontaneously, so they would always be present in the population, but a substantial percentage of them should be eliminated at each generation. However, investigation does not bear out this expectation. Confining our attention to Africa and Asia, where most of the work has been done (the disease also occurs in some Mediterranean areas and the West Indies), we can see that there are large areas where the gene occurs (usually in a single dose, of course) in 15–20 per cent of the population (Fig. 15). Within these areas there are even communities with averages up to 40 per cent.

The reason it is possible to measure the frequency of the gene in the population (thus getting the information given in Figure 15), although the character is recessive, is because, even when the chromosomes are heterozygous for the mutant (i.e. inherited from only one parent), its effects can under some circumstances be detected. The bearer does not show the disease, but under conditions of low oxygen pressure, produced by very high altitudes, or artificially in the laboratory, the blood cells will show some sickling.

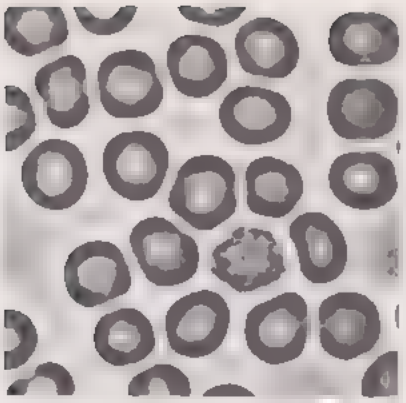
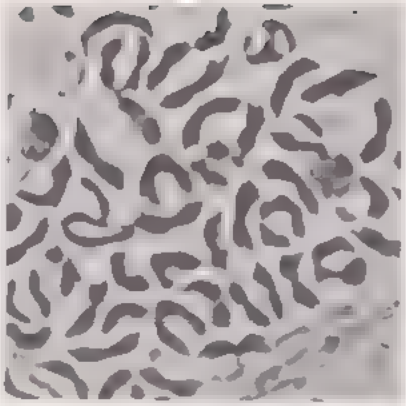


Figure 14 (a) *Hb.S.Hb.S High O_2 level*



(b) *Hb.S.Hb.S Low O_2 level*

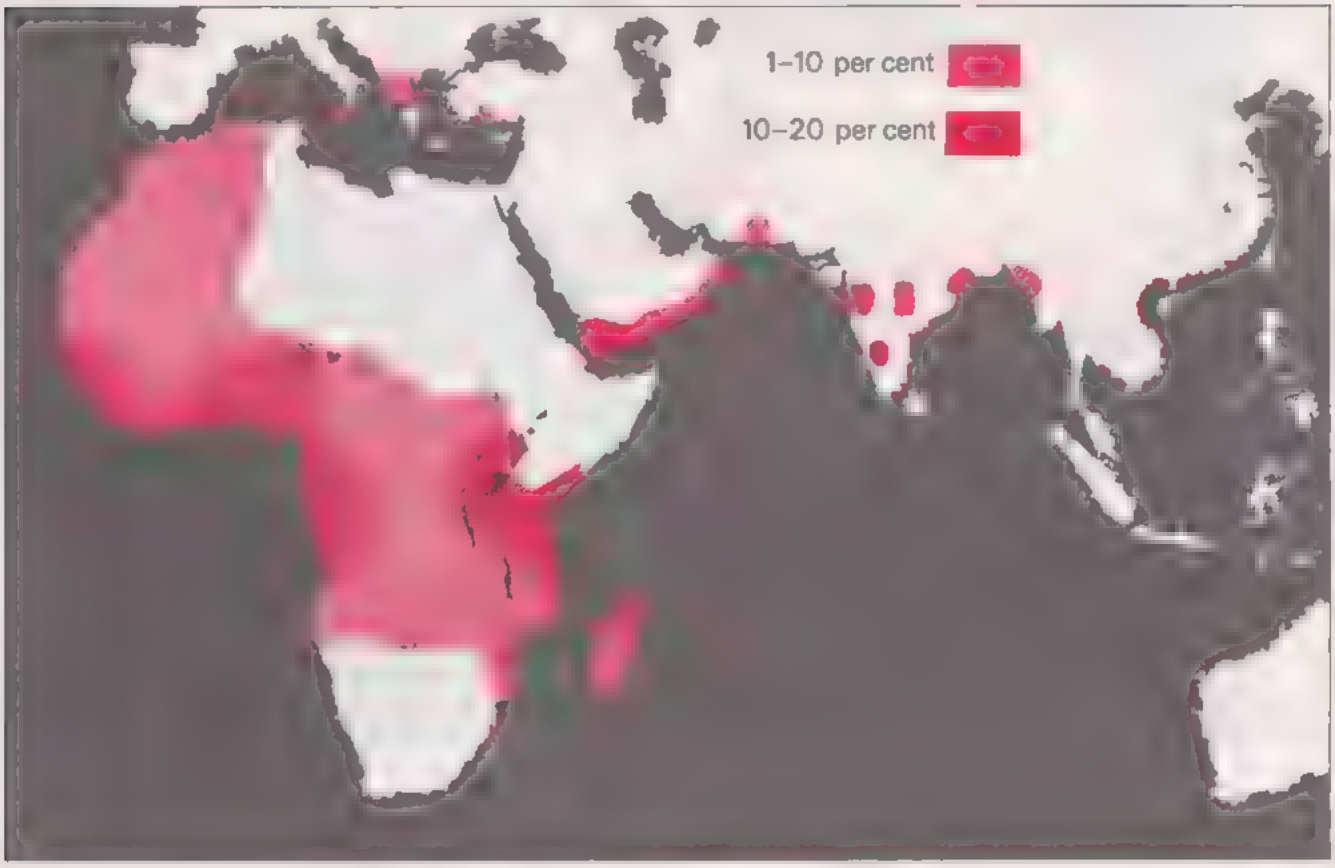


Figure 15 Map showing the distribution and frequency of the sickle-cell gene.

Normally, unless the bearer is a mountaineer, there is no visible effect on the blood of the phenotype.

Clearly the gene is not being removed from the population at the rate (16 per cent per generation) which should apply at this frequency. Not only does direct observation suggest that the levels are fairly constant, but the mutation would have to have occurred with quite incredible frequency sometime in the recent past to account for the levels observed on the basis of selective disadvantage. Thus, in spite of what we have said about the debilitating and lethal effects of the disease, we can only conclude that in some way the Hb.S gene confers a considerable positive selective advantage over the normal Hb gene, enough to offset the losses of genes at each premature death of a phenotype from anaemia.

Can you think at what stage this advantage could possibly be felt? The clue lies in what we said in the section of genotype phenotype relationships section 19.3 and above, when discussing the heterozygous condition.

Thus, investigators concluded, as you may have done, that possession of a single Hb.S gene conferred some selective advantage that outweighed the harmful effects of the double dose. A possible answer may be found on the map (Fig. 16) showing the areas with a high death rate from malignant tertian malaria. Compare this with the known incidence of the Hb.S gene. Malaria is a disease caused by an organism which lives and multiplies in the red blood cells.

Figure 16 *Distribution of malignant malaria.*

This suggested to the investigators that a single Hb S gene might provide resistance to the malarial parasite, perhaps at the stage when it is normally living inside the red blood cells and destroying the haemoglobin. Their research has largely confirmed this. In these areas, malaria is almost always present and the child mortality from it is high. (If the children survive it they normally acquire considerable immunity.) Children with a single Hb.S gene were shown to have a 25 per cent better chance of surviving attacks of malaria than those with the normal genes. There is further confirmation. There are regions within the malarious areas where the conditions do not favour the mosquitoes which transmit the malaria. In these regions, malaria is not much of a problem and, as expected, the Hb.S gene is a rarity.

It emerges, therefore, that selection is not acting to remove the genes causing sickle-cell anaemia, as might have been expected. They are indeed removed when homozygous, but this selection is outweighed by their selective advantage when heterozygous, due to the effect of malignant malaria.

In summary:

(1) Hb.Hb	(2) Hb.Hb.S	(3) Hb.S.Hb.S
normal phenotype susceptible to malaria	*normal phenotype resistant to malaria*	*anaemic phenotype usually dies*

'Malarial selection' favours (2) but, as the frequency of the gene rises, (3) will appear more commonly, and die off. Therefore a balance is reached, in which the frequency of the gene will vary considerably with the intensity of the 'malarial selection' pressure.

If this interpretation of the situation in Africa and elsewhere is correct, our knowledge of population movements would suggest that the sickling gene has been in the population in this sort of balance for thousands of years.

If you changed the environment of one of these populations with a 20 per cent frequency of Hb.S, in their gene pool, so that malaria was no longer a major cause of death, would you expect:

(a) that the frequency, now so well established would remain roughly constant;

(b) that the frequency would decline at each generation by a predictable amount, until it is eliminated altogether;

(c) that the frequency would decline at each generation by a predictable amount to a low level where the rate of elimination equalled the rate of production of new Hb.S mutants?

(c) is what would be predicted.

The first stages of this have in fact been demonstrated. American Negro slaves were imported from West Africa in large numbers between 250–300 years ago. If the frequency of the Hb.S gene in the original population is assumed to have been not less than 22 per cent, and it is allowed that this figure would be reduced by interbreeding to around 15 per cent, it is possible to predict to what level the gene frequency should have fallen today.

In the absence of any selective advantage, after twelve generations the deaths from anaemia should have reduced the frequency to 9 per cent (which would result in 1 per cent actually showing the disease). This is, in fact, the frequency found among US negroes at the present time. On the other hand, West Indians in Britain (with a very recent change of environment) show an incidence of 18–20 per cent. This fact was not appreciated until several West Indian patients in the UK collapsed whilst having teeth removed under anaesthesia, because of sickling of the red blood cells. (The dental anaesthetics used may lower the 0_2 level in the blood.)

Warfarin resistance and selection

It is interesting to speculate on whether a similar situation of 'balanced' selection pressures may not be operating in the case of the Warfarin-resistant rats mentioned in section 19.4.2 above. You may recall that the mutation which conferred resistance left them physiologically weaker than normal sensitive rats, when it appeared in the homozygous condition. This fact emerged when efforts were made to keep resistant rats in the laboratory to investigate the physiological basis of the resistance. The rats which were homozygous for this mutant gene died very easily and appeared to be deficient in vitamin K. It will be interesting to see if further work reveals that malaria resistance in man and Warfarin resistance in rats are similar illustrations of the same evolutionary situation: namely a mixed

population maintained by the balance of two potentially conflicting selection pressures.

19.6.5 The stabilizing effect of selection

An important point which has emerged by inference several times above is that natural selection does not necessarily act to produce change; it may well produce stability. Indeed, in the short term, it is far more often doing the latter, and this is of the greatest importance.

We have seen that, in fact, most mutations are likely to be deleterious to a greater or lesser extent. The genes thus produced will be eliminated by natural selection. The speed at which this happens will depend on whether the gene is dominant or recessive, and whether the new trait that the mutant gene gives rise to is merely slightly less suited to the environment than the original, or is downright lethal.

In a theoretical environment which remains constant, once a population has become well adapted, it would be reasonable to suppose that there will be no adaptive change, and that selection will be concerned *only* with maintaining stability by eliminating less fit variants. In practice it probably never comes to this. Environments are not constant. If the physical surroundings remain constant, there will still be changes in either the predators, the prey, the parasites or the competitors. Evolution appears to be a continuous process, so no environment is likely to be by-passed indefinitely. There are a few which have changed little, such as parts of the deep sea and some islands, and these may support such well-known 'living fossils' as the coelocanth, a fish which has scarcely changed since the Cretaceous, and the tuatara, a very primitive reptile with a bone structure which has remained unchanged for 170 million years (see also *Understanding the Earth*). The tuatara has now to be vigorously protected from competition with modern mammals. On the other hand, a few forms are so efficient that they seem to be able to survive considerable changes in the environment without having to undergo much more adaptation themselves—for instance, many insects appear much as they did in the Cretaceous period, 120 million years ago.

'living fossils'

So the pattern is usually one in which selection maintains the stability of a well-adapted population within a reasonably constant environment. Whenever environmental changes do occur, however, selection will lead to adaptive change in the population, provided always that variants are produced in sufficient numbers. If, however, *too many* variants are produced, the stabilizing effects of selection will be removing too many 'unfit' individuals, with the result that the population will decline. Natural selection has of course favoured the optimum balance, and herein lies the risk of raising the mutation rate in human populations by increasing the doses of radiation they receive. A population with too little variation may die as the environment changes, but one with too much may die in the existing one.

Much of this stabilizing selection will occur before the variant sees the light of day. For example, major genetic changes may result in the death of the zygote during the first few divisions—in the case of mammals before it has even implanted in the uterus. Structural changes of various kinds may put the embryo at risk at a later stage and spontaneous abortion may occur. It is estimated that twenty per cent of the human eggs successfully fertilized do not in fact survive until normal birth. This process is sometimes called internal selection, but it is the same process that we have been discussing all along. Fitness to obtain nourishment from the wall of the uterus is no different in principle from fitness to digest the mother's milk once out in the world.

19.7 Speciation

19.7.1 Species

We have seen how selection of spontaneously occurring variants may lead to changes in a population, sometimes quite major ones, but in none of the examples given did we claim that the changed population no longer belonged to the same species. Yet it is obviously central to the theory of evolution by natural selection that such processes *do* lead to the formation of completely new species, and it is quite logical to suppose that this indeed happens. We have not yet demonstrated it for two reasons, one of time-scale and one of definition. To merit the description of a new species, a population will have to undergo a whole series of changes, usually involving both structure and behaviour, which with most organisms will involve a much longer period of time than we have been considering. In addition, we are brought up against one of the most difficult points in any discussion of speciation, namely 'what constitutes a species?'

The taxonomic view of species

The science of taxonomy, the classification of organisms into groups with clearly defined similarities and differences, really owes its beginning to the work of Linnaeus (1707–1778). He ranked organisms according to the degree of anatomical similarity, with species as the basic fixed units. These corresponded to the popular 'kinds' of animals—a 'cat' an 'elephant' and so on—and he gave each a Latin name of two words. The first word was the *generic* name and the second is the *specific* name. Where species had obvious affinities, for example cat and puma, he grouped them in the same genus. This is still the system, the cat being *Felis maniculata*, the puma, *Felis concolor*. Minor varieties or races might be denoted by adding a sub-specific name after the specific one. For example, the English wren is *Troglodytes troglodytes troglodytes*.

species

As our knowledge has grown, the number of the hierarchical ranks originally suggested by Linnaeus have to be increased, but the principle remains the same. A complete classification of a species will involve placing it within a system of twelve steps or ranks, denoting degrees of affinity. However, some of those ranks are met with very much more frequently than others. The smallest grouping of species (one which indicates those species with a large number of characters in common) is into a genus, as indicated above with reference to some of the cats.

Another example is modern man, whose specific name is *sapiens*. He belongs to the genus *Homo*, which he shares with a number of fossil ancestors (mentioned again in section 19.8). This genus shows close affinities only with other extinct genera, and it is grouped with these into the family *Hominidae*. This family is grouped in turn with two others, which contain the gibbons, chimpanzees, orang-utan and gorillas, into a superfamily, the *Hominoidea*. This process is continued until man's ranking with all other living organisms is described.

This system of classification has been extended to embrace over 1.5 million species, distinguished primarily on anatomical features. These include:

Vertebrates	Mammals	about 4 000 species
	Birds	about 9 000 species
	Reptiles and Amphibians	about 6 000 species
	Fish	more than 20 000 species
Invertebrates		1 050 000 (including 850 000 insects)
Plants		about 400 000
Unicellular organisms		more than 75 000

It may be deduced from these numbers that the process of describing and determining these species is one for the specialist. Indeed, such a detailed knowledge of the anatomy and life-history of an organism and its relatives is required for this purpose, that the field of expertise of a taxonomist may be a very narrow one.

This approach to the definition of species is one of several, and possibly it is a rather more static one than that adopted elsewhere in this Unit. Nevertheless it is a view of great practical importance. In the practice of both pure and applied science, it is necessary to be able to refer to a group of organisms in such a way that everyone concerned is clear as to the precise limits of this group. For example, it may make a great deal of difference to know which of two rather similar insect pests is attacking a crop. Differences in some stage of their life cycle could call for completely different control measures.

Furthermore, the taxonomist's species do really exist. These species may live side by side and yet remain distinct, as, for example, do 200 species of fish of the genus *Haplochromis* in Lake Malawi (Nyasa). As you will see in Units 20 and 21, such groups live and interact with one another in predictable ways, the interacting units being discrete species.

Thus the work of Linnaeus began to bring order, precision and system into descriptive biology, but his system was to some extent arbitrary, and misleadingly simple. This was because it was based simply on structure, and it also assumed that species were immutable. The Creator had made all organisms at the beginning and they stayed that way. Thus the similarities between the cat and the puma were due to a whim, albeit an Almighty one. (Presumably on that day all the carnivorous animals were being run off to a similar pattern.) Species were obviously the fundamental unit because they were clearly distinguishable and unchanging. Today a classification has to represent something more than anatomical similarity, it must indicate common origins and family relationships. Nevertheless, Linnaeus' judgement was such that most of his species still stand, even though they may be grouped into different genera.

From what you know of evolutionary mechanisms would you expect to be able to classify all organisms into distinct species in Linnaean terms?

No; once you begin to collect organisms from all over the world, you find some intermediate populations, having some of the properties of one species, but with others usually ascribed to a related species.

Both Lamarck and Darwin, who assembled immense collections from regions all over the world, often found intermediates they could not reasonably say clearly belonged, on anatomical grounds, to either one or other of two related species. This confirmed their belief in the view that species evolved from other species. The problem of definition is therefore acute. An intermediate can be described as a 'race' or sub-species of one

or other of the species, but at what point must we elevate this race to a species? If biological evolution is the dynamic process we think it is, then 'species' must be a dynamic concept and we must accept that a species exists only for a period in time. Obviously then, it is a term of convenience (even if an essential one for the rational study of biology), and the lines of demarcation will be to a large extent biologically arbitrary. What is more, what constitutes a satisfactory specific character may vary with the field of study of the biologist concerned. To an anatomist or museum-based taxonomist, physical characteristics such as size and details of anatomy may be the most important; to someone studying organisms in relation to their environment, it may be their habits; to a geneticist, it is the frequency of particular genes in the genotype.

There is, however, one common factor involving all these approaches to the problem of the distinction between race and species. This is whether or not the race or variant population interbreeds with the 'original' or 'main line' population. If it does, we would expect to find a continuous physical gradation from the 'norm' of the original population to that of the race. If they meet and mate under natural conditions, their habits and behaviour must also still be closely related. Lastly, and most important, if they are exchanging genes they are effectively 'diluting' the differences which selection may be increasing. Thus, if they are interbreeding to a significant degree, it is reasonable to consider that the variant population is still only a race of the original.

The other side of this coin is that the major step in species formation is when a race becomes reproductively isolated from the parent population.

19.7.2 Gene pools

In section 19.4, we described the evolutionary unit as the 'freely interbreeding population', the unit on which selection can be seen to act. We can adopt a narrow genetical view of this unit, disregard the phenotype altogether and consider only the genotypes. In fact, it is convenient to go still further, and regard the population only in terms of the gametes it produces (the haploid germ cells), a few of which will fuse with one another to form the new generation. This rather theoretical entity is called a *gene pool*; in the terms in which we defined a population, every gamete (or gene combination) in a gene pool should have an equal chance of fertilizing any other.

In 1909, a geneticist, Hardy, and a mathematician, Weinberg, produced a simple equation which predicts the distribution of genes within such a gene pool, under ideal conditions. The Hardy-Weinberg equation forms the basis of almost all modern population genetics. It states, in effect, that for any pair of genes controlling a character, one dominant and one recessive, their relative frequencies in the gene pool, whatever they may be, will, in the absence of factors which modify gene frequency, remain constant from generation to generation. The assumption is made that the population is sufficiently large for sampling errors to be ignored.

The equation gives a precise answer to the problem exercising the early evolutionists, including Darwin: namely, why sexual reproduction does not lead to increasing uniformity. The proportions of variants are maintained with a random distribution—some individuals homozygous for one gene, a majority heterozygous, and some homozygous for the other.

The main modifying factor is selection, and where a gene is favoured or discriminated against, the equilibrium will be shifted. In addition, if a gene has a high mutation rate, or if significant numbers of phenotypes carrying it migrate in or out of the population, the equilibrium will be disturbed. However, the Hardy-Weinberg prediction will only apply where there is

reasonable intermixing. As soon as part of the population ceases to exchange genes freely with the rest, there is a probability that divergence will take place.

Reproductive isolation within gene pools

isolation of gene pools

This can come about through various circumstances, and the effects are progressive. Gene flow from one side of a large population to the other may be interrupted by geographic factors—i.e. 'barriers' such as mountains, deserts or water—or simply by the discontinuity of the population in an area. If disease, or fire, or a predator effectively wipes out the population in an intermediate area, recolonization may take time. During this time, the 'outlying' population will exchange few genes with the rest (Fig. 17). If conditions differ substantially, the effects of selection on the relative frequencies of the genes in the two groups may become apparent.

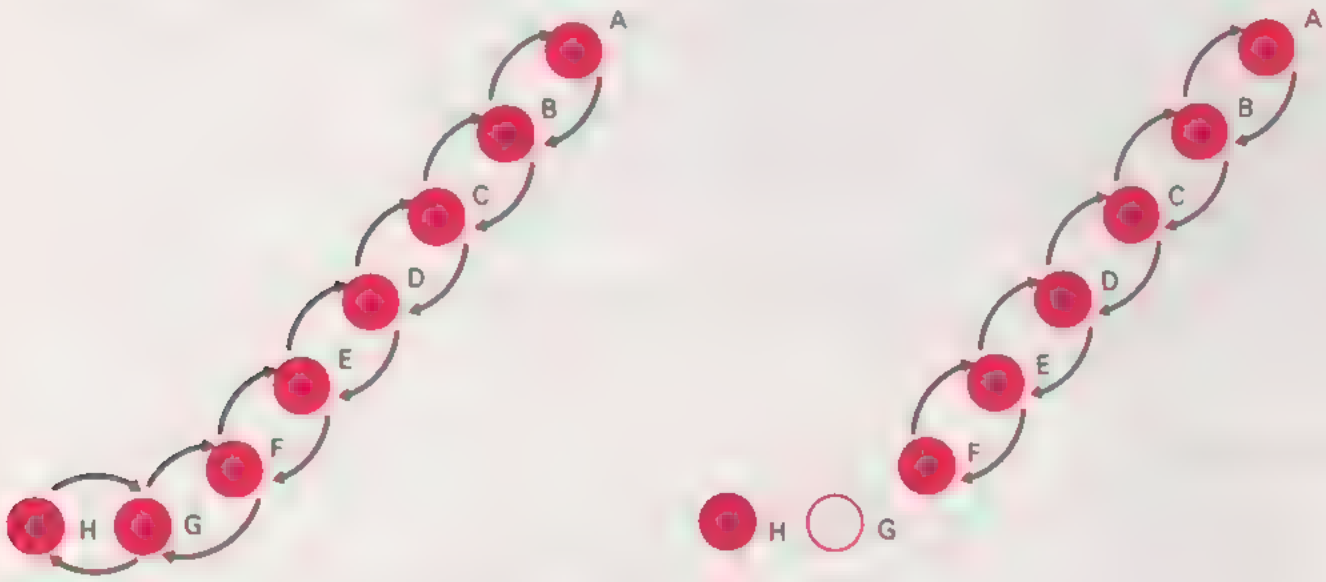


Figure 17 Interruption of gene flow across an interbreeding population.

Any of these factors will obviously encourage race formation, or, where it is already present, slow down exchange between race and parent population. The crucial factor, however, is that once the differences are there, reproductive isolation will tend to increase, thus further increasing divergence.

An actual example of this is provided by the distribution of the breeding ranges of the thirty-four 'races' of the American Song Sparrow *Melospiza melodia* (Fig. 18). Where the bird occurs in mountainous regions or on offshore islands, distinct races have become isolated from one another, each sometimes having only a few square miles of territory. Where these geographical barriers are less pronounced, a single race may have a territory of thousands of square miles, across which gene flow can occur relatively unimpeded.

In Unit 21 you will find an example (Darwin's finches) where this process has proceeded a stage further.

Isolation maintained by behaviour

Small differences in the markings of birds will reduce their attractiveness as mates to members of the original population. (See the offprint with this Unit.) Changes in song patterns may have the same effect. If an animal changes its feeding habits it meets and mates less frequently with the main population. (Races of mosquitoes apparently sharing the same environment may never meet for practical purposes if one group develops day

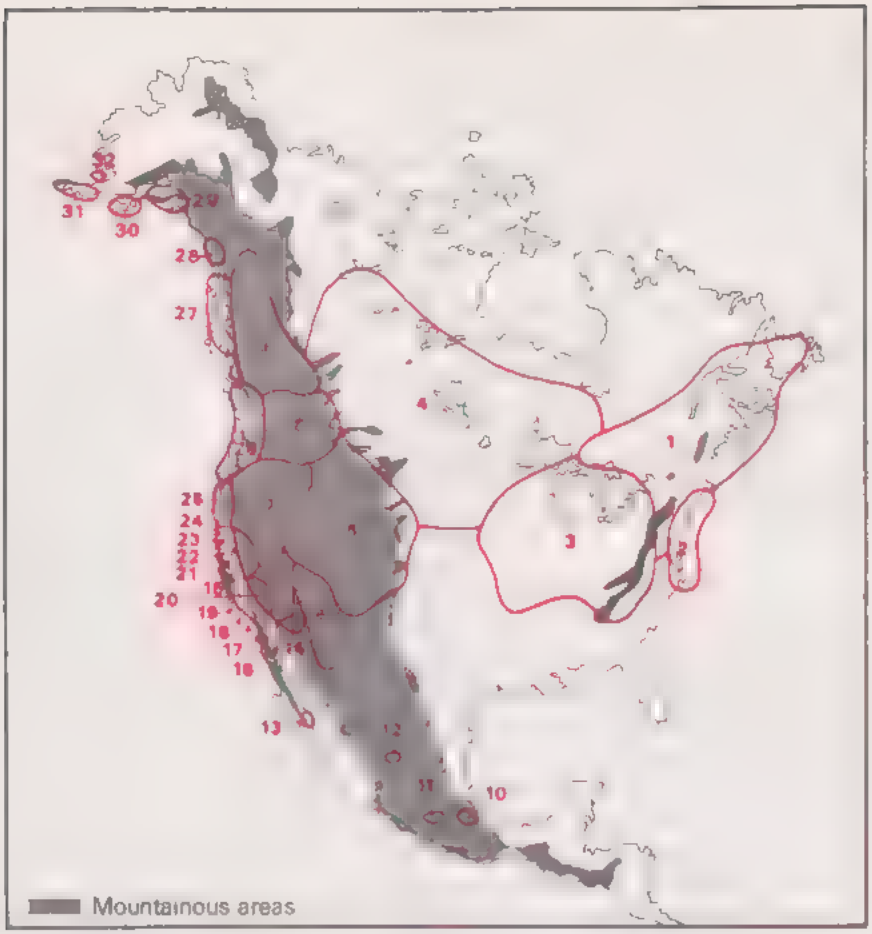


Figure 16 *Distribution of races of* **Melospiza melodia** *in North America, showing main geographical features.*

flying and feeding habits and the other evening or night flying ones) These, and many other small differences, may effectively isolate one race or part of a gene pool from the rest, and selection will increase the divergence. Thus they are no longer part of the same pool. But are they a new species?

The 'touchstone' usually employed in deciding whether a variant population constitutes a race or a separate species, is to see if they will interbreed with the 'parent population' and produce fertile offspring. Where the genetic differences have become marked, the offspring (called hybrids) are normally infertile. This is due to various factors, often the inability of maternally and paternally derived homologous chromosomes to 'recognize' one another and pair up correctly at meiosis Many such cases of hybrid sterility are well known The mule (horse × donkey) is a case in point. It must be bred from horses and donkeys each time, not from other mules. In this case, reproductive isolation is complete; no genes will be exchanged between horses and donkeys Sometimes it is only a question of degree. Domestic cattle can be crossed with American buffalo, yaks and other relations. The hybrid bulls are sterile, but the cows are fertile, and will backcross with domestic bulls, buffalo or yak. In the case of two particular species of *Drosophila*, there is merely a lowered fertility in the hybrids, but this, coupled with other isolating factors (they have rather different habitats within the same area; the males prefer the females of their own species; and they are most active at different times of day), means that under natural conditions they do not exchange genes. Thus, in this case at least, four partially isolating factors operate cumulatively and lead to complete natural isolation. So even failure to interbreed is not a simple and absolute criterion to use to distinguish separate species.

hybrids

Although the two species of *Drosophila* referred to above can in fact still exchange genes (though they seldom do), they are considered as discrete species. Similarly the races of *Melodia* can interbreed, but usually do not, yet they are considered to be a single species. If you, yourself, could sit on a California hillside for a hundred years, watching these birds, you would probably observe only minor changes in the race. On your return to civilization, however, it is possible that you would find that you had witnessed the evolution of a new species.

From what you have read in this section, then, you will not be surprised that it has not been possible to say that 'species A1 has been shown evolving from species A by a process of natural selection'. There is much evidence from which we can infer the truth of such a statement, for example, the evidence from micro-evolution, and the observable changes in gene-frequency in populations. But our own time scale, and that of the development of the theory itself, makes it unlikely that we could ever claim to have actually witnessed the event.

We suggest that at this point you should read the set text, which for this Unit is an offprint from the journal *Scientific American* of October 1967. It is an article by Dr. N. G. Smith describing work he has done on the reproductive isolation of four closely related species of gulls, nesting in overlapping territories. In addition to providing an illustration of one of the points made above, it is interesting as an example of the type of experimental situation with which a biologist may be confronted. You may feel that travelling between Arctic cliffs in a canoe, carrying tranquillizers and a paint box has nothing in common with manipulating electronic devices in the laboratory. Yet it has, the logical processes, the formulation and testing of hypothesis, analysis of the data and checking the significance of the figures, are all common to both. The main differences probably lie more in the temperaments of the investigators.

We have offered you a few simple questions which you may find useful. In two of them we ask you to comment critically on some of the statements in the article. (An attitude we hope you will adopt to all our material.) If you feel that *you* would have written them differently, bear in mind that this is an article from a magazine with a mixed readership, not the publication of detailed results in a specialized journal.

What was the basis of Smith's claim that pair formation depended on female initiative?

See Answer 9, p. 77.

Which of the following factors does this work show to be *irrelevant* to successful copulation in established pairs of gulls?

(a) Soliciting behaviour of the female.

(b) Wing-tip pattern of the female.

(c) Eye contrasts of the female.

(d) Wing-tip pattern of the male.

(e) Eye contrasts of the male.

(f) State of development of the male's testes.

See Answer 10, p. 77.

On p. 7, column 3, paragraph 1, Smith says his original working hypothesis on why pairs stayed together before egg laying was that 'the main component of the pair bond was the attachment of the individuals to one another'. Would you consider this a useful hypothesis?

See Answer 11, p. 78.

On p. 7, column 1, Smith refers to natural selection in the following terms. 'To avoid mixed pairings, then, selection favoured dark-eyed individuals where Kumlien's gulls, etc. . . .'

(a) Do you consider it legitimate to think of natural selection in these terms?

(b) It is suggested on p. 101 and p. 102 (bottom of column 1 and top of column 2) that natural selection is maintaining differences in the populations which tend to discourage interbreeding and the formation of hybrids. What underlying (but unstated) assumption must Smith be making about the hybrids, if his claim is true?

See Answer 12, p. 78.

Do you feel that this work has established a major cause of reproductive isolation between the four species concerned in terms that should satisfy a scientist? If not, what further questions would you suggest Smith should have tried to answer?

See Answer 13, p. 78.

19.8 Human Evolution

This is also the topic of the radio programme of this Unit. Human evolution provides an interesting and slightly unusual example of selection at work. If you can remember as far back as Unit 1, you will recall we suggested that man was remarkable for the development of his brain, and that a significant increase in brain size had occurred since the time of his ancestor of half a million years ago, *Homo erectus*. It is striking that in man's general bodily form there has been almost no sign of specialization over more than three million years, other than changes in the pelvis and foot allowing upright walking. In fact, man is built on all-purpose lines. He can run, crawl, dig, climb and swim; he has excellent vision (day and night), reasonable hearing and a fair sense of smell. (No other animal can do all of these things as well as man, though each of them can be done very much better by one specialist or another.) But while the body has not increased greatly in size since *Australopithecus*, a man-ape of a million years ago (whose average height was about four foot, six inches), the cranial capacity has risen by 300 per cent. (See Table 5, Fig. 19, p. 55, and the Radio Broadcast Notes.)

increase in skull capacity

Table 5 Cranial capacity in human evolution

	Capacity (in cm^3)
Australopithecines ('man-apes')	450–550
Homo erectus erectus (Java man)	770–1 000
Homo erectus pekinensis (Peking man)	900–1 200
Homo sapiens neanderthalensis (Neanderthal man)	1 300–1 425
Homo sapiens sapiens (recent)	1 200–1 500

What then is the selective advantage which has resulted in this change? Clearly having a larger head has no advantage *per se*; it merely requires larger hats.

No simple direct relationship between skull capacity and mental capacity has been shown among living humans, and of course we cannot prove that modern man has greater intellectual potential than Java man. However, there is evidence that, between species, more brain cells in the cerebral cortex make for a more extensive memory, and also, as you will remember from Unit 1, a proportionate increase in the brain/body ratio leaves more nerve cells free from the 'autonomic' control of the body (see Unit 18) and available for 'higher' functions. It is also known that memory systems seem to involve a very large number of cells and that relatively enormous areas of the brain are involved in controlling the fine manipulation of the hands, tongue and lips.

It is reasonable to assume, therefore, that the expression of this increase was the ability to use, and later make, tools and to develop sophisticated speech. The advantages of both attainments in controlling the immediate environment are immense and obvious.

Tool manufacture was a characteristic of the man-apes who inhabited Olduvai Gorge (Tanzania) a million years ago. The ability to make a tool for the job is a great advance on the habit of using something that happens

to be at hand, as do some apes and birds The skilled use of tools has come with the development of a thumb which can be used in opposition to the fingers, a necessity for fine manipulation.

Speech confers great advantages over any simple communication system to a hunter in the planning and co-ordination of food catching, but again it calls for huge numbers of extra nerve cells Thus it is reasonably safe to say that man's evolution has centred round the evolution of his brain, selection favouring the skills made possible by an enlarged mental capacity.

All the fossil specimens of *Homo* that are thirty thousand years old or less (e.g. Cromagnon man, Fig. 19) are 'modern' in all respects, that is to say they are not structurally distinguishable as a sub-species from ourselves.

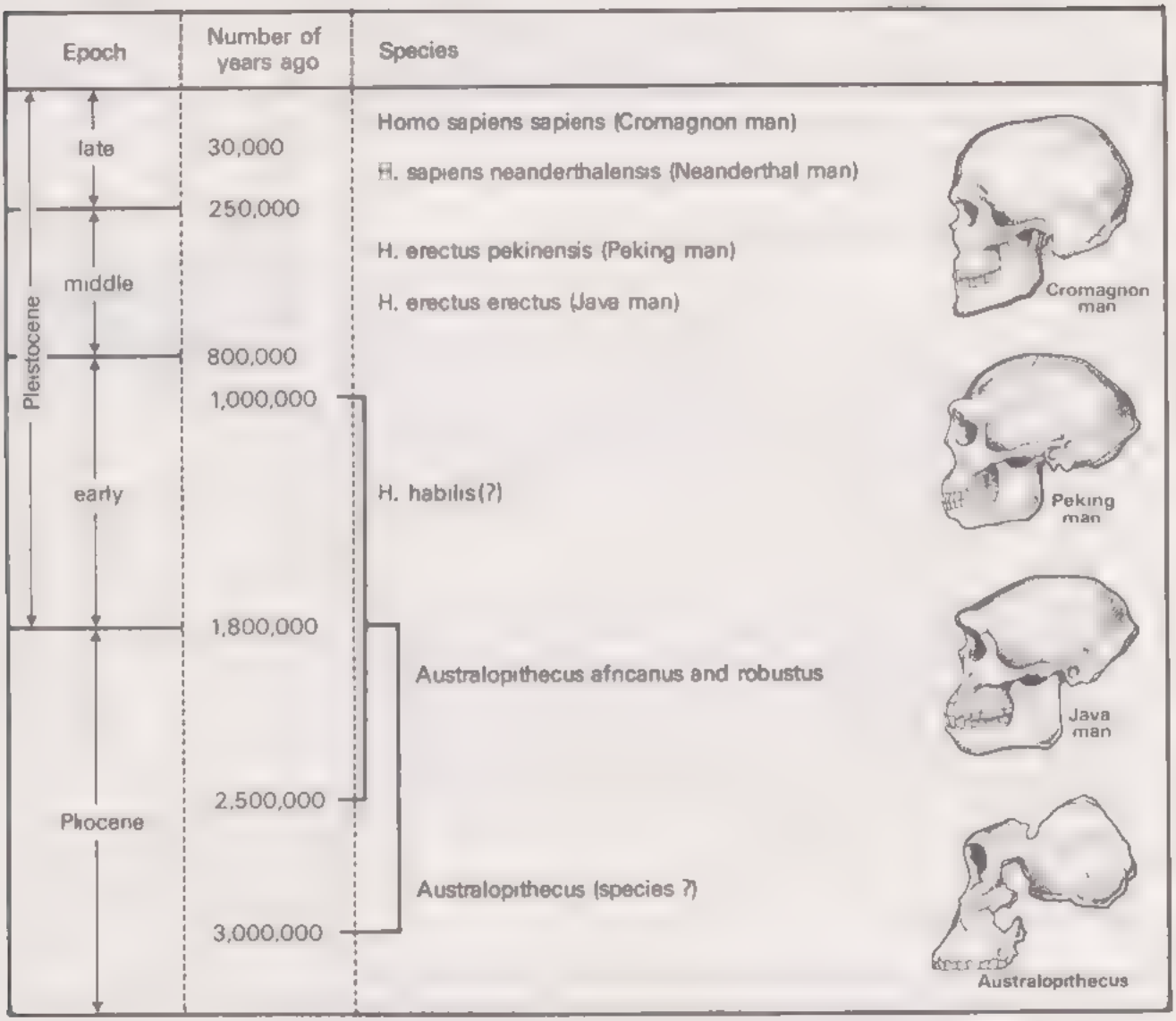


Figure 19

There is much evidence that their culture involved complex rituals and elaborate art, as indeed did that of some earlier groups, yet their basic 'economics' had apparently remained almost unchanged for several *million* years. Their stone implements were similar in function to those of *H habilis* and the species of *Australopithecus* resident in Olduvai Gorge between two-and-a-half million and one million years ago. Like these latter species, *H sapiens* was still a hunter and vegetable gatherer (It is estimated that hunting accounted for about 25 per cent of his food, plant collecting about 75 per cent.) This sort of life requires a basically nomadic habit, and the land will only support a relatively low density of population.

The first evidence of any fundamental change in the way of life of *Homo* comes from what is sometimes called the 'New Stone Age', and, in particular, in the Middle-Eastern populations of about 8–10 thousand years ago. They show, for the first time, evidence of the domestication of animals and the cultivation of crops. This change is an essential one for the formation of larger human communities with a basically 'fixed abode'. It can be argued that human development remained merely an extension of this way of life until the Industrial Revolution. It is interesting to think that we may be witnessing the start of the third major change, the 'space age', to have taken place since the rise of *Australopithecus* between two and four million years ago.

19.8.1 Is man still evolving?

As the history of species goes, *Homo sapiens* is young, and it would be surprising to find that he had stabilized his gene pool by eliminating all the modifying factors discussed in earlier sections. Indeed, what we know of human genetics suggests that selection may be acting quite fiercely to change some genes.

Can you think of an example which we have mentioned?

Sickle-cell anaemia.

The Hb.S,Hb combination is not favoured in the USA, or anywhere else where malaria is being controlled, and it is being eliminated from such [illegible]

The fact that we are living in an increasingly controlled and artificial environment merely means we must adapt to it, as we have had to adapt to changed conditions in the past. For example, genotypes sensitive to drugs such as penicillin and barbiturates (and they are not uncommon) will be eliminated as unfit, where previously they were neutral. The laboratory rat has changed so that it is unfit for life in the wild; but it is not decadent, merely adapted, as it is far better suited to life in the laboratory than is the wild rat. We may be moving towards a similarly enclosed existence, and selection may adapt us to it.

Of course, the situation is not always as clear-cut, since adaptation can only take place through reproductive fitness. For example, a 'virtue' in modern living may be the ability to resist new kinds of pressures, those caused by overcrowding, noise, pollutants and the more sophisticated social stresses. If, however, people have completed their reproductive lives *before* retiring into a mental hospital or dying of bronchitis, then selection cannot act. At the moment we really do not have the information to say whether those particularly susceptible to such things are reproductively as fit as the rest of the population or not.

19.8.2 Selection and intelligence

A most interesting example is the effect of selection on intelligence. Even if man's present dominance of his environment stems from his increased mental capacities, it does not follow that what is now needed for the benefit of mankind is greater intellectual advance, though many would assume that it is desirable. Nevertheless, the question remains: 'is selection favouring intelligence now, as we assume it did in our past?'

The first pitfall in assessing this is that we have no satisfactory definition of intelligence—it depends very much on the context in which it is being measured. Thus it can equally be said to be manifest in acts of character judgement, prudent cowardice or computer design. We commonly tend to measure the so-called Intelligence Quotient of a person by comparing his score in an intelligence test with a supposed national average performance of 100. The problems associated with this method are numerous, not the least being that what an intelligence test measures is the ability

to do intelligence tests, and many feel that there is only a marginal correlation between this ability and intellectual attainment. This has been nicely illustrated with rats. A group of highly 'intelligent' rats were produced by rigorous selection over several generations for their ability to run a maze. Thus, within four or five generations, they showed a marked shortening of the time taken to learn the maze, compared to that taken by controls or their own great-grandparents. However, it emerged that these faster-learning, or 'more intelligent' rats were only quicker at learning mazes. They showed no significant improvement in other types of learning and problem-solving situations. Thus 'intelligence' is not really an acceptable entity for study; it is necessary to specify a particular skill. But, in the absence of a better criterion, we still tend to use the skill of 'intelligence test passing' as our yardstick.

The second pitfall lies in a situation which is most obvious in the human organism: the interaction between the genetically-determined potential and the effects of education or training. We saw something of this when considering the athlete in the section on genotype and phenotype (p. 24). The potential mental capacity may be expected to be determined genetically, as with all other physical traits. However, the early environment seems to play a major role in deciding to what extent this potential will be realized. If this environment does not provide an adequate pattern of stimulation, development may be permanently retarded, even to the point where speech is difficult. Thus, although intelligence tests are designed to avoid testing formal learning, they cannot avoid measuring many environmental influences.

Having given these warnings, what of the results? There is a *prima facie* case for thinking that intelligence should be on the decline, at least in the Western world. It has been widely recognized, since the end of the seventeenth century, that throughout the West, and possibly China, the more successful members of the community have had fewer children than the norm. This is true whether 'success' is judged by social class, income or education. Not only would these people be expected to have higher than average intelligence quotients, but they would be expected to provide above average environments for their children. Measurements of I.Q. support this expectation. Thus, in terms of reproductive fitness, those with higher I.Q.s are at a disadvantage. This has been estimated to produce an anticipated drop of 1–4 points on the I.Q. scale per generation in the average level of the population; a fearful prospect.

There is very little evidence to decide if this expectation is being fulfilled. Only two really widespread comparisons over a sufficiently long time interval have been made, one on 88 per cent of all Scottish school children, with a fifteen-year interval (1932 and 1947) between measurements, and one on American soldiers in the First and Second World Wars, with an interval of some twenty-five years. The results of these showed that the scores on the more recent tests were, if anything, slightly higher; an unexpected and apparently anomalous result.

Assuming the information we have given you as to the fertility of those with higher I.Q.s is correct, can you account for this result?

One possible reason is education. We said that I.Q. tests measure education to some extent, and for the population as a whole this improved vastly in the periods under consideration. Another is that better diets can be shown to improve I.Q. and the national diet can be shown to have improved.

Thus it appears that if it is true that intelligence is being selected against, this is being more than compensated for, at the moment at least, by improvements in the general level of education, including perhaps at a pre-school level.

At the other end of the scale, another factor may be at work which could have a stabilizing effect on population I.Q. Recent work suggests that

marriage between low-grade mental defectives may produce only slightly more children than those between people of very high I.Q.

The highest fertility is to be found among the mediocre intelligence levels So selection may, in fact, tend to eliminate the very stupid as well as the bright, thus maintaining something near the *status quo* (i.e. 'stabilizing selection').

Table 6 Mean number of siblings of each inmate of a mental institution

Parents	*Cases observed*	*Siblings (mean)*
Superior × normal	9	2.89
Normal × normal	798	4.72
Normal × dull	196	5.45
Normal × feebleminded and dull × dull	113	4.52
Normal × imbecile and dull × feebleminded	54	3.82
Dull × imbecile and feebleminded × feebleminded	24	3.58

Table 6 shows how many full brothers and sisters (siblings) there were of each inmate of an institution for mental defectives. The inmates have been grouped according to the I.Q.s of their parents.

All of this is sufficiently unproven to provide a fertile field of argument and clashing faiths. Current trends in human evolution are very difficult to distinguish, but evolution of some kind is undoubtedly taking place. What we *can* say is that today *Homo sapiens* is a single species, made up of several races all of whom share a large number of genes. These races are now exchanging more genes than they were even 500 years ago, when limited local colonization was probably the main cause. Today, almost every country has representatives of almost every other one living in it and to some extent intermarrying within it. Thus, in addition to cultural exchanges and changes, modern mobility is providing new genetic cocktails for new and existing selection pressures to work on. This will inevitably mean more change.

19.9 Summary of the Unit

1 To account for the great diversity of species, and the complexity of the adaptation they show to their environment, it is necessary to accept either that they were created as they are by a Divine Being, or that some process of organic evolution has taken place. The first interpretation is not examined in detail, but the second forms the substance of the Unit.

2 (a) If species have evolved, then it is clear there must be heritable variation as well as the continuity stressed in Unit 17. One of the ways the genotype of an organism may change is by mutation. This involves the re-arrangement or substitution of one or more nucleotides on the DNA of the chromosome. This change in the sequence of nucleotides results in a change in the polypeptide chains encoded in that part of the DNA. This will lead to changes in the proteins of the cell and thus visible phenotypic changes (e.g. phage resistance in *E. coli*, or its inability to manufacture tryptophan, defective haemoglobins in man, colour changes in *Sordaria* spores).

The main cause of naturally occurring mutations is not certain, but it is probably a chemical factor or factors within the cell. Many exogenous chemicals will raise the mutation rate, as will irradiation and, in some species, temperature rises. The timing of mutations appear to be quite random, but the rate varies between species and between genes within a species.

(b) Heritable variation also arises through sexual reproduction because of the fusion of gametes from different parents and because the haploid gametes are produced by meiosis. Meiosis involves the random assortment of the homologous pairs of chromosomes prior to division and, in many cases, crossing over. Thus the existing genetic material of a population is remixed and reshuffled at each generation.

3 Darwin laid emphasis on natural selection as the main factor leading to adaptation and evolution, with the inheritance of acquired characters and mutation as lesser factors. He postulated that overproduction within populations would lead to competition and that this would result in the selection of some variants more frequently than others, thus leading to a change in the population. Experiments can be designed to show whether selection acts on spontaneously occurring variation or on change actually induced by the environment.

4 Several examples of the micro-evolution that has taken place in recent years are considered. These include the selection of strains of pests and disease organisms which are resistant to the control measures used against them, and the deliberate selection by man of crops and stock showing certain characteristics.

5 Animals live in limited, interbreeding populations, and each genotype may be considered as contributing to the 'gene pool' of this population. The frequency of the genes in the gene pool will remain roughly constant so long as the members can interbreed, except where pressures such as selection act to change the frequency of a gene. If the members cease to interbreed freely, one part of the pool becoming isolated from

the next, then selection or other forces may cause divergence between the main pool and the isolate. Such isolation may become permanent if the phenotypic expression of the divergence even further discourages the exchange of genes.

This may lead to the formation of new species, or just distinctive races or sub-species. The borderline between species and race is difficult to define, but is usually drawn on the basis that definite anatomical differences together with a *de facto* failure to exchange genes will justify distinction at specific level. There are many exceptions to this generalization however.

6 Human evolution and the effects of selection on the human genotype are briefly considered.

Appendix 1 (White)

Glossary

ACHONDROPLASTIC DWARFISM A hereditary abnormality in which the limbs are much foreshortened, although the head and trunk remain normal.

ANTIBIOTIC A substance, produced by a living organism, which diffuses into its surroundings where it is toxic to some other species. Closely related substances can now be synthesized and go under the same general name.

GENOTYPE The genetic constitution of an organism (i.e. the particular set of pairs of genes in each cell of an organism). (See Unit 17.)

HAEMOPHILIAC One suffering from the hereditary disease, haemophilia, in which the ability of the blood to clot is lost or impaired. In serious cases even minor internal damage can lead to fatal haemorrhage.

HETEROZYGOUS Having two different genes (e g the normal gene for a character and its mutant) at the corresponding points on a pair of homologous chromosomes.

HOMOZYGOUS Having identical genes at the corresponding points on a pair of homologous chromosomes.

PHENOTYPE The sum of the characteristics manifested by an organism, as contrasted to the genotype. (See Unit 17.)

SPINDLE A body which forms in the cell during division, and takes part in the distribution of chromatids to the daughter nuclei. The chromosomes become arranged around its equator. It appears to be a gel, largely composed of longitudinally arranged protein fibres which are synthesized shortly before division. The movement apart of the chromatids (or chromosomes) may be due to the contraction of some of the fibres.

SULPHONAMIDES Sometimes called sulpha drugs. A group of organic compounds containing the group $SO_2 . NH_2$ as its derivatives. The first drug which could be used effectively to kill bacteria without killing the patient. First introduced in the 1930s and still widely used.

Appendix 2

Factors Affecting the Mutation Rate

1. Chemical Mutagens

A whole range of chemical substances will induce mutation, and are therefore said to have a *mutagenic* action, or to be *mutagens*. Such chemicals include hydrogen peroxide, urethane, manganous chloride, nitrous acid, mustard gas (dichloro-diethyl-sulphide) and many others. It may be that many of these mutagens act by causing replication faults or by changing the template. There is good evidence, too, that they may act to cause breaks in the DNA molecule. This can result either in the deletion of a whole segment, or in a re-arrangement of whole genes, a fact which can be demonstrated by working out the linear sequence of genes along a particular chromosome, thus making what is called a *linkage map*, before and after treatment with a mutagen.

mutagen

Some chemical mutagens can definitely be shown to act by causing chromosome breaks and other aberrations. Colchicine, for example, inhibits formation of the mitotic spindle and this causes the chromosome number to double at each division. Mustard gas can be shown to cause chromosome breaks in the fruit-fly *Drosophila*. These breaks are generally repaired immediately, but often with deletions, or with whole lengths of DNA moved to a different location. Such mutations should perhaps be termed 'chromosome mutations' to distinguish them from the gene mutations discussed elsewhere.

No evidence which has been offered so far proves that *every* change of a nucleotide produces a functional mutation; quite possibly it does not. It is worth remembering that there is not a one-to-one relationship between mutable sites on the DNA and any one amino acid of a protein. The *E. coli* enzyme *tryptophan synthetase*, which is the one referred to in the main text, illustrates this. It is possible to isolate a back-mutation in the tryptophan-requiring strain, in which the 'defective' amino acid is replaced by the correct one, restoring the activity of the enzyme. Investigation shows, however, that this is sometimes due not to a reversal of the original mutation, but to another change on an adjacent site. It is therefore not possible to say, in the present state of knowledge, to what extent every nucleotide will matter functionally.

tryptophan synthetase

The situation is further complicated by the fact that mutation may well involve many more than one nucleotide. On occasions, a mutation may involve the breaking and rejoining of whole chunks of DNA.

Simple substitutions of a nucleotide are likely to arise from time to time as errors in replication. As Unit 17 demonstrated, replication is a very accurate process, the probability of error in the incorporation of a new nucleotide may be as low as 10^{-8} or 10^{-9}, but this is still appreciable. Such errors must arise through failure to form the proper hydrogen bonds, perhaps with the accidental pairing of adenine with guanine instead of thymine. These errors can be produced experimentally.

Quite a lot is now known about the effects of nitrous acid on the template. For example, it appears that it de-aminates adenine, replacing NH_2 with keto groups, forming hypoxanthine. This will then pair with cytosine, rather than with the original thymine. Similarly, cytosine is de-aminated to uracil, which bonds to adenine instead of guanine.

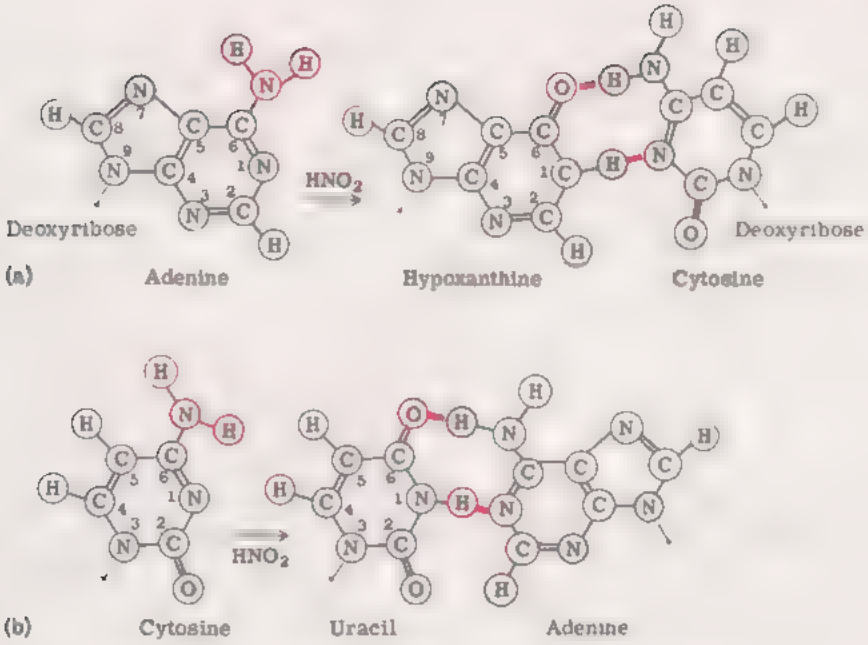


Figure 20 The effect of nitrous acid in causing amino-acid substitutions.

2. Ionizing Radiation

Ionizing radiations (Unit 35), in this context meaning short wavelength electromagnetic radiation (e g X-rays and γ-rays) and accelerated particles with high energy (e g. cosmic rays), have long been known to cause mutation. In 1927, Muller showed that the progeny of *Drosophila* irradiated with X-rays showed more mutations than progeny from non-irradiated parents (Table 7).

Table 7

Muller's experiment in the induction of mutations by X-ray treatments in chromosomes of *Drosophila melanogaster*.

(The T_4 dose is twice as large as the T_2 dose.)

Experiment	*Number of chromosomes tested*	*Mutations observed*		
		Lethals	*Semi-lethals*	*Visibles*
Control	198	0	0	0
X-rays (T_2)	676	49	4	1
X-rays (T_4)	772	89	12	3

He later showed that the frequency of mutation is directly proportional to the dose of X-rays received, measured in *rads*. A rad is a unit of *absorbed* radiation (in tissues or any other material) (Fig. 21). It corresponds to 100 ergs/gramme of material.

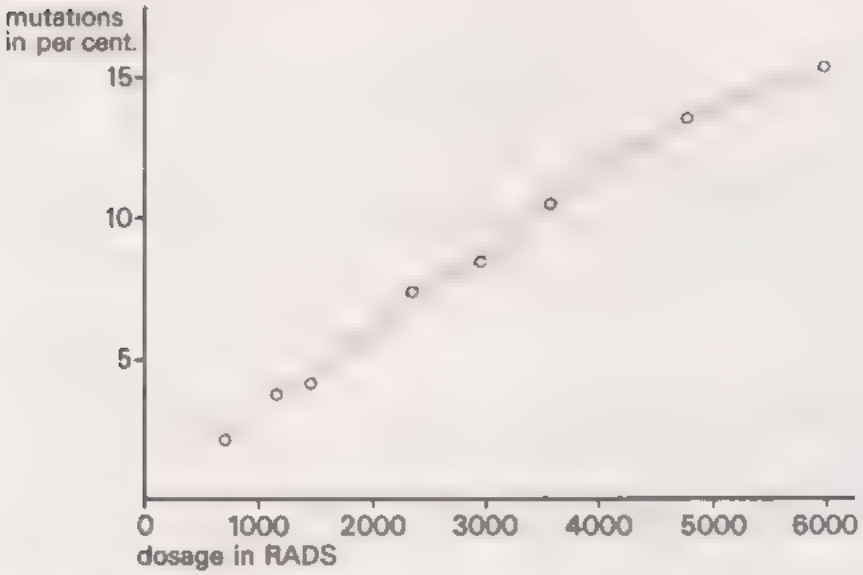


Figure 21 The effect of X-rays on the mutation rate of Drosophila.

It made no difference whether this dose was received over a few minutes at a high intensity, or over months or even years at a very low intensity; the number of mutations remained proportional to the total number of rads received. However, this simple relationship does not always hold true Where the doses are large enough to damage the normal metabolic processes of the cell, it may make a difference whether the whole dose is given in one exposure or in several. Thus, in mice, it can be shown that a single dose of 1 000 rads produces fewer mutations than two doses of 500 rads given 24 hours apart. One of the reasons for this seems to be that where the single larger dose is given many of the mutated cells are killed and therefore do not come to light. There is also some evidence that the surviving cells from one dose are more mutable than the original cell population, when given another dose within certain time limits In general terms, however, the larger the number of rads received, the greater the number of mutations produced, although there are complicating factors.

This obviously has great bearing in assessing the importance to humans of the radiation received from various sources, natural, medical, military or commercial. It is not true to say that there is a 'threshold' level of radiation below which additional mutations will not occur. We can only say that we are (or are not) prepared to put up with a particular avoidable increase of the mutation rate in the population.

It has also been shown that all radiation in this range has an equivalent effect if compared dose for dose The mutagenic effect of such radiation does not seem to vary significantly over quite a wide temperature range, which is interesting in view of the effect of temperature on the spontaneous mutation rate (see below) Some other factors *do* vary the effect of a given number of rads, however, notably the amount of oxygen in the tissue The lower the oxygen level the less frequent the mutations.

It is not certain exactly how the mutations are produced Visible chromosome breaks may occur, in addition to changes within a gene, but it is not known whether the production of a mutagenic chemical (for example, hydrogen peroxide, which might account for the effect of the absence of oxygen mentioned above) follows a 'hit' close to a relevant hydrogen

bond, or whether the mechanism is even more direct. What is apparent is that it is a very localized occurrence. A particular dose or intensity of radiation does not produce a particular type of mutation throughout the tissue involved, presumably the damage occurs in a molecule only in the immediate vicinity of where radiation is absorbed and ionization occurs. In this context, it is interesting to note that ultra-violet light, which has a longer wavelength (of the order of thousands of ångströms), also produces mutations. However, different parts of the ultra-violet spectrum have vastly differing mutagenic abilities which precisely parallel the degree of absorption of the respective wavelength by nucleic acid The higher the absorption, the greater the mutagenic effect.

It is, therefore, to be expected that ionizing radiations will cause a significant number of mutations in a population, whether the source is natural —e.g. cosmic rays, background radiation from granite rocks, etc.—or man made. It is doubtful, however, if this is the prime cause of 'natural' mutation Certainly, in the case of *Drosophila*, it has been calculated that a fly would have to receive about 60 rads of radiation in four weeks (a fairly generous estimate of its generation time) to account for the observed rate of mutation, if this were the only cause. It is likely that in this time the fly would, on average, receive only about 0.05 rads, which would account for only 0.1 per cent of the observed mutations. However, insects are unusually resistant to radiation, so such calculations may not necessarily be relevant to mammals.

3. Temperature

Muller, in 1928, using the same technique as he employed to assess the effects of X-rays (see above) on mutation rate, also examined the effects of temperature.

Once again using *Drosophila*, he found that populations kept at higher temperatures showed a higher mutation rate. A rise in temperature from 19° C–27° C (8° C) increased the mutation rate by a factor of 2.5. Other experiments on different species have confirmed that a usual figure is an increase by a factor of 2–3 for a rise in temperature of 10° C.

This might be taken as an indication that multimolecular chemical reactions are involved in a normal mutation, as this is approximately the rate at which temperature rises effect such reaction rates However, the increased mutation rate produced by irradiation does not vary with changes in body temperature, which suggests that such a reaction is not involved in the latter case. This is further evidence that 'spontaneous' mutations may not be caused by radiation.

Appendix 3

1. Polyploidy and Variation

Another important source of genetic variation arises from a failure of normal division. This failure may come about if the spindle does not form correctly. The result of this is that one of the daughter cells gets all the chromosomes and the other none, leading to a diploid gamete and a zygote with one-and-a-half times the diploid number ('triploid'). If both gametes are diploid the zygote will be 'tetraploid'. Any organism which has more than the two normal diploid sets of chromosomes is said to be 'polyploid'. Triploidy and tetraploidy are two particular cases of polyploidy. Polyploidy is rare in animals, and usually results in a non-viable zygote. Thus the triploid human cell, a photograph of which is shown in Figure 22, is very unusual. However, one extra sex chromosome is not so uncommon, even in humans. Where the extra chromosomes do not form a complete set, the condition is not usually called polyploid, but it may nevertheless have a profound effect on the phenotype. For example, a human being with an additional chromosome of pair number 21 suffers from the condition known as mongoloid idiocy, or 'mongolism'. The chromosomes of one such person are illustrated here, also those of a 'feminized male' with one extra X chromosome, XXY instead of XY (male) or XX (female). In plants, however, polyploids are much more common—it is believed that about thirty per cent of all the flowering plants are polyploids. It used to be thought that this invariably lead to gigantism—for example, the giant sunflower is a polyploid variant of the normal one. However, although the individual cells generally *are* larger, the plant as a whole may not be, sometimes just some part of the plant useful to man may be enlarged, and this category includes a number of plants of great importance to man, such as some species of wheat, maize, tomatoes, potatoes, New World cottons and cultivated sugar cane. All these are polyploid versions of wild ancestors. For example, the wild American cotton has 26 chromosomes, 13 pairs, and is a normal diploid; but the form with the useful fibre is tetraploid, with 52 chromosomes forming 26 pairs. There is evidence to suggest that this has arisen from a cross between two different species of diploid cottons, forming what is generally called a *hybrid*. Hybrids are, of course, not necessarily polyploid; there are many diploid hybrids (see section 19.7), but these are normally sterile. Polyploid hybrid plants tend to show extra vigour and size: consider, for example, the difference between normal and giant sunflowers, wild wheats and oats and cultivated ones. Thus in plants polyploidy may constitute an important source of variation from an evolutionary point of view. Polyploidy may also occur at mitosis, if the chromosomes duplicate but the spindle fails to form and separate them. This effect can be produced in the laboratory by treating dividing cells with the substance colchicine. In multicellular animals, however, mitotic polyploidy is not very likely to be transmitted, and from this point of view is of less interest here.

triploid

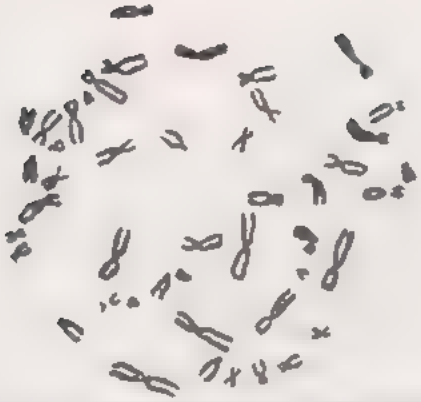
mongolism

hybrid

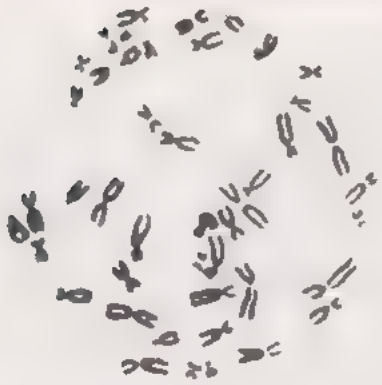
Relative importance of different sources of variation

We have considered the main sources of genetic variation in organisms: first, mutation, some of the causes of it, and its frequency; secondly, the effects of meiosis and sexual reproduction on the re-assortment of chromosomes and the re-combination of genes within the chromosomes; finally, a brief look at increases in the chromosome number, polyploidy. The relative importance of these factors in producing changed genotypes will

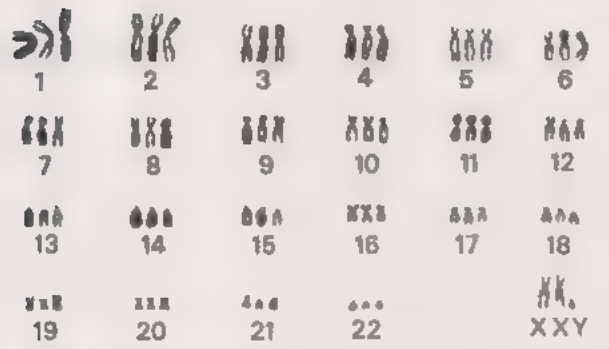
Appendix 3



1 Chromosomes of normal Human Male (displayed in sequence below)

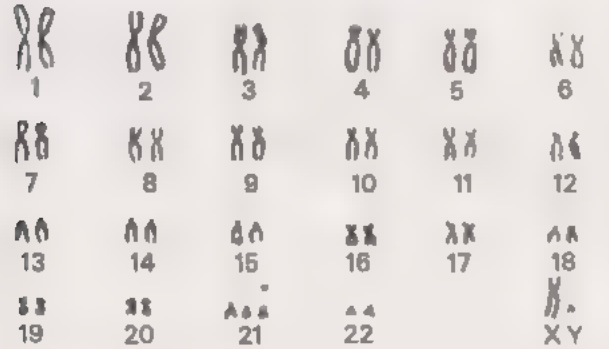


2 Same but for Female



3 Human Triploid (spontaneously aborted)

4 Feminised Male (Klinefelters syndrome, sterile)



5 Mongoloid Idiocy (male)

6 Sexually immature Female (Turner's syndrome, sterile)

Figure 22 Photomicrographs of stained human chromosomes, as they appear in the cell, and displayed in sequence of homologous pairs. Asterisks mark the abnormal arrangements in 4, 5 and 6.

vary enormously, and it is not worth generalizing However, meiosis followed by fusion of the haploid gametes certainly leads to the most frequent changes in genotype—effectively every time in fact These changes will, in principle, be small ones, because they are re-combinations of characters already being carried in the genotype of the species But for some reason they should—once again in principle at least all be viable characters. In contrast, mutation can produce quite new genes, but when an organism is an enormously complicated system of interrelated and interdependent structures and reactions (and most are), arbitrary changes are most likely to be harmful to a greater or lesser degree When an undergraduate from a residential university celebrates, and afterwards replaces two of the semi-conductors in a computer with a tomato, or other randomly chosen component, it would be surprising to find that a useful change had been made, and though just the simple deletion of one small part might not affect the performance of the entire machine, it is likely to make it slightly less efficient, not more so It will only be rarely that the operator will try out the change next morning and find that the computer works better than it did before.

It is difficult to compare polyploidy with the other changes, as it is so unclear why in some cases it results in 'more of the same', which is often good for the species, whilst in many organisms it merely results in genetic disaster. However, it is interesting to take note of the Evening Primrose, *Oenothera lamarckiana*, in this connection. It was as a result of studying this plant in 1901 that de Vries first coined the term 'mutation' in a genetic context, to describe the sudden changes he observed in the organism in his garden. It produced: a 'mutant' *Oenothera gigas*, of increased size, a dwarf form, *nanella*; several colour changes, and so on. Modern research has shown, however, that only about two of these new forms were caused by what *we* call mutants. The others were caused by a variety of genetic changes. *Gigas* is a straightforward tetraploid version of the parent plant (*lamarckiana*), having 28 chromosomes instead of 14. Others had 15 chromosomes, as one was present three times instead of twice *Nanella* was the result of a complicated piece of genetic behaviour, which will not be elaborated upon here, stemming from the fact that *lamarckiana* is a type of hybrid, and it produces two very different types of gametes, which leads to curious results when it fertilizes itself. Thus the variations were being produced by mutation, polyploidy and an unusual consequence of meiotic assortment of chromosomes. In this particular case the three processes were of comparable importance.

2. Challenges to the Darwinian View

Even after the rise of genetics as a science had provided massive support for a modified Darwinian view, situations were demonstrated which some scientists considered to show support for a form of Larmarckian mechanism. Try to interpret the following examples according to each theory in turn, and see which you find the most convincing.

1 A Russian agriculturist called Lysenko, claimed during the 1930s to have established beyond all doubt the inheritance of acquired characters in crop plants, particularly wheats. In one of the best known of his experiments, he took a widely used variety of wheat, which was giving only a modest yield in the field, and bred it for several generations under test conditions, with good protection, water supply and large quantities of fertilizers, etc. The yields under these conditions rose sharply in successive generations. Then it was tried for a year under normal field conditions and gave a higher yield than it had originally. He claimed that by producing heavier yields under ideal conditions, he had actually 'conditioned' the wheat to transmit this ability to subsequent generations.

Can you think of one explanation for his results in Darwinian terms—and how you would test your interpretations?

Under ideal conditions, with all the normal hazards removed, the heavy cropping strains would be favoured, as they produce more, and larger, seeds. Thus Lysenko may well have merely selected the genotypes which gave the heaviest yields per plant, disregarding any other possible selection pressures. When the wheat was first returned to the field, the predominance of the heavy cropping genotypes led to higher yields. These genotypes were not necessarily those giving high resistance to rust fungus, drought, high winds, etc., which is why they were not dominant in the original population. Thus, on Darwinian theory, he would have merely selected for those genotypes most suited to an ideal environment and *not* those best suited to the open field. On this basis, we should expect a very high mortality among these genotypes in the field, and an increase in the proportion of seeds derived from the smaller, hardier plants. This appears to have been the case; after one season the proportion of heavy cropping plants fell away, as did the total yields.

Yet the Lysenko story is of more than merely passing interest, for it also provides an example of the relationship between science and wider issues. In the context of the agriculturally impoverished Soviet Union of the 1930s and 1940s, Lysenko's ideas were seized upon eagerly by those attempting to improve agricultural productivity. His opponents, classical geneticists led by the famous plant-breeder Vavilov, were forced into the defensive and many of them lost their jobs and some their lives. Lysenko's theories *had* to be right, it was claimed, and the 'normal' rules of scientific procedure and evidence were disregarded. The situation in the Soviet Union has since been 'normalized' once more, Lysenko's opponents have been rehabilitated—sometimes posthumously, while Lysenko himself continues to run a minor institute. The dispute divided the scientific world both in and out of Russia, for it was concerned not merely with the *facts* of the particular matter (and the complexity of the relationships between the organism, its development, genetics and environment are still only partially understood today)—but still more importantly with the relationship between the ideas and paradigms (Unit 1) of a scientist and the social environment in which he works. We return to some of these issues in Units 33 and 34.

2 In 1949, a curious transformation was shown to occur in the unicellular animal *Paramoecium aurelia*. If a suspension of some thousands of paramoecium is injected into the blood stream of a rabbit, the rabbit reacts as if these were germs (as it would with any foreign protein) and manufactures a substance in the blood which will destroy them. After this, samples of that rabbit's blood, if added to a culture of the paramoecium, will destroy it. However, it will only kill paramoecium of that particular strain. Other strains, say from another pond, will not be affected, unless the rabbit has been injected with them also. So we can

say that the different strains of *Paramoecium aurelia* fall into several different categories with respect to rabbit blood. The strains breed true with regard to these categories—the descendants of a culture in category 'A' are also in that category. However, if you take a culture of paramoecium in category 'A' and subject to a *weak* solution of the blood of a rabbit previously injected with that strain, so that the paramoecium are not actually killed, you find that the culture has changed category to one of the other groups; and it breeds true.

It appears that the nature of the substance produced by the rabbit is determined by bodies in the cytoplasm of the paramoecium. But in a paramoecium of, say, category 'A' not all the bodies are appropriate to this category; merely a great majority of them. It also contains different proportions of the other bodies, those which would, if in the majority, make it belong to one of the other categories. Thus, if a paramoecium in category 'A' is attacked by a weak solution of the blood of a rabbit containing the 'anti-A' substance, the 'A' bodies are destroyed. If the animal survives, one of the other groups of bodies multiplies up and becomes dominant; thus the animal has changed categories, and it will breed true for its new category. Does this seem to you to be a genuine case of the inheritance of an acquired change?

Self-Assessment Questions

Section 19.2

Question 1 (*Objective 1*)

Which of the following (a–f) are true and which false?

1 Crossing over is:

(a) the exchange of genetic material between homologous chromosomes;

(b) the mixing of genetic material from two different species;

(c) the process by which homologous pairs of chromosomes come to be randomly orientated during the first meiotic division.

2 Variation in the genotype will not be expected:

(d) in a clone of micro-organisms;

(e) between identical twin mammals;

(f) in the offspring of a mating between identical twin mammals.

Question 2 (*Objective 1*)

Which do you consider the clearest and most explanatory of the definitions of the gene below?

(a) A gene is the smallest part of the DNA of a chromosome which will determine the structure of a complete polypeptide in the phenotype.

(b) A gene is a discrete particle or segment of the chromosome which determines a trait or character in the phenotype.

(c) 'What we shall call genes are special, separable and therefore independent "conditions", "bases" or "materials" that are present in the germ cells, and which determine at least many properties of the organism' (paraphrased from Johannsen, 1909).

Sections 19.2 and 19.6

Question 3 (*Objective 2*)

In various parts of this Unit you have been given information about, or asked to consider, various aspects of the disease sickle-cell anaemia. Now turn to the list of objectives and consider objectives 2 (a–g). Which of these objectives may be illustrated by what you know of sickle-cell anaemia, and why?

Question 4 (*Objective 4*)

Potentially, sexual reproduction will be expected to confer certain advantages on a species, when compared to asexual reproduction. What is this advantage (a) and how does it arise (b)?

Section 19.3

Question 5 (*Objective 1*)

Select the best definition of phenotype:

(a) the arrangement of genetic material on which selection can act,

(b) the physical or 'bodily' expression of the genes or genotype;

(c) the structure resulting from the interaction of the physical expression of the genes or genotype with the environment.

Section 19.5

Question 6

Write not more than half a dozen sentences which summarize what you consider to be the important points made in section 19.5.

Section 19.6

Question 7 (*Objective 2(e)*)

Write in not more than half a dozen sentences what you consider to be the important points made in section 19.6.

Section 19.7

Question 8 (*Objective 7*)

Join the ranks of the illustrious by attempting to define 'a Species'.

Section 19.8

Question 9 (*Objective 8 (c)*)

Draw up headings for an essay on the theme that 'Change in the human genotype is likely to be an important factor in future human evolution'. *Do not attempt this until you have listened to the radio programme of this Unit.*

Question 1

(a) true, (b) false – this is hybrid formation; (c) false; (d) false, mutation may occur, (e) substantially true, as they are derived from a single egg. However, there may have been mutations in individual cells of the body, but these would probably be undetectable. (f) a non-starter. The parents are genetically identical, and therefore of the same sex, so they are unlikely to have any offspring.

Question 2

(b) has the advantage of simplicity, but does not explain much. (a) is the most precise description, and in line with the thinking in this Foundation course (c) is the first definition, and still holds good, but suffers from the same disadvantages as (b).

Question 3

2(c) The substitution of valine for glutamic acid in position six of the β chain of the haemoglobin of the blood, a consequence of a single mutation.

2(e) Possessing the sickling gene in the heterozygous condition may be considered an adaptation to life in a malarious area.

2(f) The maintenance of the Hb.S gene in 20 per cent of the population in some malarious areas (but not elsewhere) provides a clear example of the effect of conflicting selection pressures.

2(g) It provides such an example both in its frequency in malarious areas, and its rapid elimination from groups which have migrated to non-malarious areas.

Question 4

(a) It gives rise to greater *variation* than could be expected from asexual reproduction. This increase in the variety of genotypes should increase the ability of the species to adapt when acted on by differing selection pressures.

(b) 1 Because *two* individuals contribute genes to each offspring.

2 Because in gamete formation there is rearrangement of gene combinations due to the independent assortment of the chromosomes and crossing over.

Question 5

(c) is better than (b). The physical expression of the genotype is indeed the phenotype, but it is only a theoretical entity if divorced from the environment. *Some* environment or other *must* have interacted with it.

Question 6

1 The development of evolutionary theory in the eighteenth century is briefly examined, and its formulation on the basis of the inheritance of acquired characters is discussed.

2 An outline of Darwin's theory of evolution by natural selection is given, and the essential ingredients of it (Overproduction, Competition, Variation) are discussed.

3 The resistance to attack by phage shown by *E. coli* is used as an example of an experimental situation allowing predictions on Lamarckian and Darwinian theory to be tested.

Question 7

1 The meaning of 'fitness' in the genetic sense is defined, and the selection of strains of organisms resistant to damaging biological and chemical agents described.

2 This is illustrated by reference to *Staphylococcus* and penicillin resistance, houseflies and DDT resistance and the resistance of strains of the rat and rabbit to Warfarin and myxomatosis, respectively.

3 Other effects of selection on populations mentioned include their ability to adapt to exploit new food sources, and their ability to adapt to use the existing food resources more efficiently (as in the selection of faster-growing domestic animals).

4 The effect on the gene pool of a population of two conflicting selection pressures is exemplified by the distribution of the sickling gene.

5 The stabilizing effect of selection is mentioned.

Question 8

Beyond what we have said in the text, we can only help you by offering you three other definitions:

1 'Speciation occurs at that stage of the evolutionary process at which the once actually or potentially interbreeding population becomes segregated into two or more separated populations which are physiologically incapable of interbreeding'.
(Adapted from Dobzhansky, 1941.)

2 'Species are groups of actually or potentially interbreeding populations which are reproductively isolated from other such groups.'
(E. Mayr, 1942, *Systematics and the Origin of Species.*)

3 A species is a group of organisms able to inter-breed to produce fully viable offspring, or which may be presumed capable of doing so should circumstances have permitted, but which if given the opportunity to mate outside the group will not thereby produce descendants able to compete successfully with members of the parental group.
(C. B. Goodhart, 1967.)

Question 9

In such an essay you must bear the following points in mind.

1 Human evolution has undoubtedly involved selective change of the genotype in the past.

2 Some changes due to selection can be *shown* to be occurring now.

3 We ourselves are changing our environment faster than we would expect obvious physical changes to arise by selection.

4 However, adaptive responses to our new environment may not necessarily *be* obvious physical ones. For example, selection for behavioural characteristics able to resist stress due to overcrowding, or for tolerance to particular contaminants in food or air, may be a very rapid process.

5 A combination of our well known technological resourcefulness and our less apparent wisdom may stabilize our environment completely, and remove the causes of competition between human beings. This could change our views on the effects of selection applied to variation as they affect man.

Answers to In-text Questions

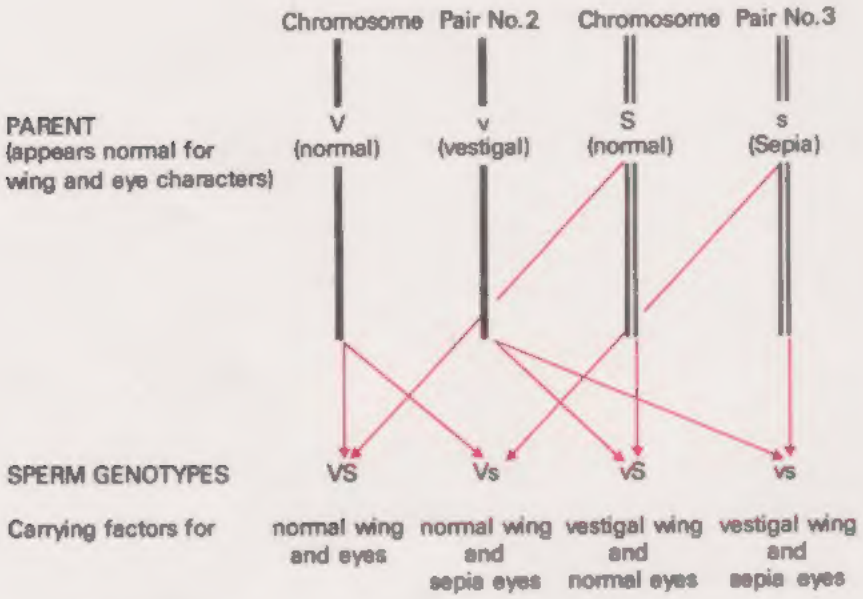
Answer 1

Sixteen genotypes:

ABCD	ABCd	Abcd	abcd
aBCD	abCD	aBcd	aBCd
AbCD	AbcD	abCd	aBcD
ABcD	ABcd	abcD	AbCd

Answer 2

There will be four different genotypes carried by the sperm for these characters:



Answer 3

Although there was a considerable overlap, the average size of the beans produced by group A were smaller than those from group B, though they were all grown under roughly similar conditions. Thus his selection was effective in separating out the two extremes of size, indicating that the difference had a genetic basis.

Answer 4

Because all the group were of the same genotype. Therefore the size differences were only phenotypic, due to environmental differences.

Answer 5

Yes, the genetic differences will remain.

Answer 6

Attack by the phage in the Petri dishes induced a change in the relevant genes of a proportion of the population, thus conferring a heritable resistance on them.

Answer 7

At some time in the course of the multiplication of the bacteria in the liquid cultures, spontaneous mutations occurred giving the possessors and their descendants immunity. These bacteria would be isolated when the whole population was attacked by phage.

Answer 8

If the change in the genotype occurs in response to the phage attack, the numbers of resistant bacteria should be roughly the same in each Petri dish. As there are enormous numbers of bacteria in each dish, and the process of becoming resistant is presumably always the same, the variation between dishes should be very small.

If resistance is a result of random mutation you would expect the numbers of resistant bacteria to vary widely from dish to dish. For example, if one mutation occurred in the culture seven generations (say three hours) earlier, then by the time liquid is poured across the jelly there will be 64 mutants, each founding a resistant colony.

Answer 9

Pair formation was unaffected by the appearance of the female, but much affected by that of the male.

Answer 10

(a) Probably very relevant in view of the statement that 'it always preceeded copulation', but these experiments do not provide evidence one way or the other.

(b) No information except in relation to pair selection.

(c) Very relevant, but the degree of relevance depended on (f).

(d) Irrelevant.

(e) Irrelevant.

(f) Relevant.

Answer 11

Not very. The whole purpose of the work was to attempt an objective analysis of the behavioural relationships between the birds. To start with 'attachment' (affection?) as a hypothesis would seem unhelpful, particularly as it is hard to see how such a hypothesis could be tested.

Answer 12

(a) No. It takes an anthropomorphic view (see Unit 1) of selection, and is teleological (see Unit 18).

(b) Selection will only favour isolating mechanisms in this way if the hybrids are less well adapted to the environment than the 'pure' species. Thus Smith is *assuming* this to be the case.

Answer 13

Yes.

His results suggest that the markings and the birds' response to them, could well account for all or much of the apparent failure to interbreed. The work does not, of course, exclude the possibility of there being other, even stronger reasons, but it is difficult to see how these could be excluded, whilst necessarily working under natural conditions.

Acknowledgements

Grateful acknowledgement is made to the following sources for illustrations used in this Unit:

A. C. ALLISON, Figs. 14, 15 and 16; BANTA TEACHING AIDS, Fig. 22; W. A. BENJAMIN INC., Figs. 2 and 20 in J. D. Watson, *Molecular Biology of the Gene*; J. & A. CHURCHILL LTD., Fig. 13 in G. E. W. Wolstenholme and M. O'Connor, *Bacterial Episomes and Plasmids*; SOCIETY FOR THE STUDY OF EVOLUTION, University of Kansas, Fig. 18.